PB82-147752

Epidemiology Feasibility Study
Effects of Noise on the Cardiovascular System

South Carolina Univ., Columbia

Prepared for

Environmental Protection Agency
Washington, DC

Sep 81

U.S. Department of Commerce
National Technical Information Service
NTIS

TECHNICAL REPORT DATA

(Please read Instructions on the reverse before completing)

1. REPORT NO.

2.

3. RECIPIENT'S ACCESSION NO.
PB82 147752

4. TITLE AND SUBTITLE
Epidemiology Feasibility Study: Effects of Noise on the Cardiovascular System

5. REPORT DATE
September 1981

6. PERFORMING ORGANIZATION CODE
363006

7. AUTHOR(S)
Shirley Thompson, Ph.D.

8. PERFORMING ORGANIZATION REPORT NO.
EPA 550/9-81-103

9. PERFORMING ORGANIZATION NAME AND ADDRESS
Dept. of Epidemiology and Biostatistics
University of South Carolina
Columbia, SC 29208

10. PROGRAM ELEMENT NO.

11. CONTRACT/GRANT NO.
68-01-6274

12. SPONSORING AGENCY NAME AND ADDRESS
Environmental Protection Agency
Office of Noise Abatement and Control
Washington, D.C. 20460

13. TYPE OF REPORT AND PERIOD COVERED

14. SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES Sent out Report with Appendix B - Annotated Bibliography and Appendix C - Review of Non-Noise Related Cardiovascular Literature

16. ABSTRACT

This reports contains a review of all world literature to date and a critical analysis of existing foreign and domestic epidemiological research on the nonauditory physiological effects of noise. In addition, the scientific literature on hypertension, cardiovascular disease, and general stress are analyzed for its applicability to noise effects epidemiology. Information and recommendations pertinent to a number of substantive and methodological issues related to the design and conduct of future noise effects epidemiology is also provided.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Cardiovascular Disease, Noise, Blood-pressure, Epidemiological Research, Long-Term Noise Exposure, Dose-Response, Stress Industrial Noise, Community Noise, Health Effects, Nonauditory Effects.		

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)* UNCLASSIFIED	21. NO. OF PAGES
	20. SECURITY CLASS *(This page)* UNCLASSIFIED	22. PRICE

EPA Form 2220-1 (9-73)

PB82-147752

EPIDEMIOLOGY FEASIBILITY STUDY:
EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM

EPA 550/9-81-103

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SEPTEMBER 1981

FINAL REPORT

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REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA 22161

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The views expressed in this report are solely those of the author and should not be construed to represent EPA policy.

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Section 1

INTRODUCTION

1.1 Purpose

The effects of noise on the health of populations are of growing concern as noise increasingly becomes a pervasive environmental stimulus, the by-product of industrialization, urbanization, modern transportation and general changes in individual lifestyles. There is strong scientific evidence that high intensity noise exposure results in hearing loss which grows more severe as exposure continues over the years. Evidence is accumulating which indicates that noise may cause adverse effects on human health other than hearing. Recent developments summarized by Kryter (1970), Ahrlin and Ohrstrom (1978), Cantrell (1979), Welch (1979), Hattis (1980), Jansen (1980), Glorig (1981) and Peterson et al (1980, 1981) suggest that the extra-auditory effects of noise may be more serious than previously suggested. These reviews further indicate that sudden bursts of either impulse or steady state noise from 75 dBA up have been found to produce various temporary changes in the physiological state of animals and man. Responses can include changes in cardiovascular blood pressure and volume, pulse rate, electrocardiographic abnormalities, respiratory rate, intestinal mobility, catecholamine secretions, urinary norepinephrine excretion, serum lipid levels, platelet aggregation and peripheral vasoconstriction. However, by far the largest body of evidence centers about the relationship between prolonged exposure to intense noise and cardiovascular effects.

Although the study of extra-auditory reactions believed to be "caused" by noise has increased significantly, contradictory results within and among animal experiments, human experiments and epidemiologic studies have obscured the relationships observed and their interpretations. It is clear that neither laboratory experiments nor epidemiologic studies in themselves can provide convincing evidence as to whether or not detrimental non-auditory health effects occur in man as the result of continuous exposure to high-level noise. Ideally, evidence from experimental and epidemiologic studies would be synthesized to present a clear picture of causation. Controlled experiments in animals and man serve to (1) direct attention to health states, physiologic responses and health behavior that may be affected by noise; (2) provide information on the mechanisms which produce the causal linkages between noise and these responses; and (3) rule out plausible alternative explanations and put disparate findings in perspective. Epidemiologic studies can best (1) provide information on long-term effects of noise on human health in populations of diverse susceptibility; (2) clarify the significance of varying levels of noise exposure on individuals living in uncontrolled environments by quantifying and comparing risks; and (3) place observed relationships in proper etiologic perspective by careful analytic control of other known risk factors of the health response under investigation.

In recent years there have been numerous symposia and reviews of the voluminous body of literature that is accumulating on health effects of noise. However, since experimental studies have predominated in this field, most of the scientific evidence has been derived from animal and human experiments of short-term exposure to noise that is not characteristic of usual human environments. Two notable exceptions are the reviews by Welch (1979) of the foreign literature on the extra-auditory health effects of industrial noise and by Taylor and colleagues (1980) with emphasis of the health effects of transportation noise. Further analysis and synthesis of the population-based data available are in order. Thus, it is the purpose of this report to review the evidence for causality based on epidemiologic data, focusing on long-term cardiovascular effects of noise exposure. Emphasis is restricted to the cardiovascular system by the complexity of the subject matter and by limitations of time and resources.

1.2 Objectives

The overall objectives of this literature analysis are (1) to evaluate the extant and relevant epidemiologic research and (2) to make recommendations as to the kind and extent of evidence that is required in future population-based studies for judging the relationship between long-term noise exposure and adverse cardiovascular effects. These objectives subsume several specific questions around which this report was organized. They are:

(1) What is the current status of epidemiologic research on hypertension and cardiovascular disease; how do these findings apply to the feasibility , design and conduct of future epidemiologic studies on noise effects?

(2) What is the overall scientific adequacy from the acoustical, medical and epidemiological perspectives of the world literature in assessing noise effects on the cardiovascular system? What are the substantive and methodologic gaps in knowledge in this research area?

(3) On the basis of the evidence, what are the implications, issues and needs for the design of future epidemiologic studies which will allow determination of the relationship between environmental noise and adverse cardiovascular system effects?

The task of addressing the above questions required operational definitions of noise and cardiovascular system effects. According to the American National Standards Institute (ANSI S3.20-1973, R 1978), noise is any undesired sound; noise is an erratic, intermittent, or statistically random oscillation. The noise may be steady, unsteady or impulsive. To meet the objectives of this project this generally accepted definition of noise was expanded to include sound other than the undesirable, that may be harmful to human health.

In the literature, the term "cardiovascular system effect" has been used to indicate any measurable physiologic change in the cardiovascular system observed as a response to noise exposure. This project applies a more strict definition and attempts to identify "effects" which are known to be detrimental and/or exhibit strong potential for initiating or enhancing adverse chronic responses. In an effort to determine the extent to which noise may act as a biological stressor, a broad range of responses were specified and are presented in Section 3 of this report.

1.3 Organization of the Project and Report

The project divides into four main phases: (1) literature search and acquisition, (2) state-of-the-art review of cardiovascular disease epidemiology, (3) critical evaluation of studies on the effect of noise on the cardiovascular system, and (4) synthesis of cardiovascular and noise effects epidemiologic evidence with recommendations for future studies.

The literature search phase consisted of several major tasks. The first was to identify and obtain key review papers on cardiovascular disease risk factors published in the last three years. Another task was to identify and access a comprehensive set of the pertinent world literature titles related to the cardiovascular effects of noise. This search included non-English as well as English language sources. Once identified, articles were obtained, screened for relevance and distributed to appropriate sources for translation. Forty-seven translations of scientific documents were obtained. Finally, nonrelevant titles were deleted and relevant titles were classified using a computerized classification system.

Phase two, the state of the art review of cardiovascular epidemiology, provided an empirical foundation for the subsequent noise related research evaluation. It summarized the current state of knowledge of pathophysiology of cardiovascular diseases and identified pathways in the pathogenic process and potential entry points for investigation of the influence of noise on the cardiovascular system. Secondly, the incidence, prevalence, secular trends and major cardiovascular risk factors were discussed with emphasis on confounding and interactive factors which must be considered in the design of future studies (see Appendix C).

Critical evaluation of the literature on the effects of noise on the cardiovascular system was the major goal of the project. This third phase consisted of four tasks: (1) development of criteria for evaluating the adequacy of the noise parameters, the quality of the cardiovascular response measures and the epidemiology methodologic aspects of the study; (2) critical review of each article by an expert review team composed of an audiologist, a cardiologist and an epidemiologist; (3) summarization of the three independent evaluations of the review team in the form of annotated bibliographic materials; and (4) discussion of the scientific merits and substantive issues from the literature with reference to the project objectives.

The final phase of the project consisted of synthesizing the information derived from the literature review and suggesting the feasibility of and designs for future epidemiologic studies on the non-auditory health effects of noise.

The results of the project are presented in the following order. Section 2 describes the conduct of the literature searches and acquisition of the reference documents. Section 3 provides a critical evaluation of the studies of noise and cardiovascular system response and Section 4 attempts to synthesize the cardiovascular and noise effects literature, culminating in a set of recommendations for future epidemiologic studies.

Section 2

WORLD LITERATURE SEARCH

2.1 The Search Strategy

The project objectives were predicated on the analysis of existing literature. Therefore, the identification and acquisition of pertinent literature, in both the areas of cardiovascular disease risk factors and the adverse effects of noise, became a major component in the study.

The project specified "world literature" so the scope of the search was essentially international, drawing on studies in many languages; it was not limited by the country of origin, by its degree of industrialization or by its relative advancement in scientific research. There was interest in research which potentially could be replicated, or at least analyzed on the basis of its adherence to scientific methodology and standards of epidemiologic research. Although the country of origin did not play a limiting factor in the identification of pertinent studies, the acceptance of this research by peers, through publication in peer review journals, government sponsorship of research, etc., was a determining factor in the search strategy.

Existing on-line bibliographic data bases, which index or abstract the majority of the world's scientific peer review journals and government sponsored research were utilized whenever possible. Additionally, printed indexes were searched for materials not identifiable on-line, such as those items published prior to the years of coverage in the bibliographic data bases.

Both for verification, and as a source of materials not identifiable through standard sources, bibliographies of classic studies were also reviewed for any potentially relevant articles and unpublished papers. These bibliographies, as well as suggestions from authorities in the field, provided a substantial source of materials; although supplementing the core of literature to some extent, they also served to validate those studies which had already been disseminated through traditional means.

The literature search, designed to identify articles, reports, etc., leading to a state of the art review of cardiovascular disease was approached first as a logical step towards determining the relevance of noise as a causal agent. This search was, by necessity, limited to specifically relevant, recent literature on cardiovascular disease. MEDLINE, or Medical Literature On-Line, a product of the MEDLARS system of the National Library of Medicine, indexes over 2,400 health sciences periodicals from around the world and is considered to be the most comprehensive and viable data base in the field of medicine. Because of the expansive coverage and controlled vocabulary, MEDLINE was chosen as the primary source for the identification of literature on the epidemiology of cardiovascular disease.

MEDLINE offers the capability of using sophisticated search techniques in addition to the utilization of the controlled vocabulary via Boolean Logic. Essentially, this allows for the identification of a maximum number of relevant citations with a minimum of false drops. The primary search strategy was to identify all relevant articles on any "cardiovascular disease" (or group of such diseases) which were termed "review" articles (E.G. EXP *CARDIOVASCULAR DISEASES AND REVIEW).

Secondly, risk factors were identified, and each of these, individually, was combined with specific forms of cardiovascular diseases to determine any relevant literature other than review articles. The primary forms of cardiovascular disease included acute myocardial infarction, angina pectoris, sudden death, hypertensive heart disease, hypertension, stroke (cerebrovascular disease), and hypercholesterolemia. Risk factors included hypertension, alcohol consumption, cigarette smoking, diabetes, cholesterol levels, psychosocial factors, age, heredity, etc. An example of this type of search would be *MYOCARDIAL INFARCTION AND *CHOLESTEROL.

Although not every combination of main or subset concepts produced a reference, enough articles were identified to give a broad-based core of literature on the subject. From this core, bibliographies of specific articles were reviewed. Any recognized "classic" study, even if it predated the proposed ten-year cut-off point, was obtained for inclusion.

Concomitant to the cardiovascular literature identification several search strategies were being formulated to identify any literature relating to possible cardiovascular effects induced by noise. The identification of this literature required a broad-based approach. For example, an article might contain the overall subject of adverse effects of noise primarily dealing with hearing loss, but certain tables and a small, although significantly important, portion of the article might deal with congestive heart failure in workers exposed to noise over long periods of time. MEDLINE was again used, and an SDI (Selective Dissemination of Information - current awareness) search was entered to provide monthly updates of recently published literature on the topic. However, because of the nature of the desired outcomes, many other data bases were employed and the searches themselves were not limited to literature solely about the topic. The general concept addressed in all searches was the possible adverse effects of noise as it related to any form of cardiovascular disease or physiological change. All types of literature were scanned, from books to conference proceedings to unpublished reports to review articles.

The MEDLARS system offered several data bases which were determined to be specifically relevant. Most significant of those was MEDLINE and its backfiles, which provided bibliographic access to the 2,400 journals from 1966 to present. Conference proceedings, chapters in books, and a few other pertinent types of literature are also included in the total data base. CATLINE provided similar access to the world's monographic health sciences literature which is housed in and cataloged by the National Library of Medicine.

SDILINE, a monthly update of the MEDLINE file, was used to provide monthly updates to the core of literature identified. The SDI search was run for a period of eight months, and during this time, two articles were indexed relating to cardiovascular effects of noise.

A CROSS search was run on the BRS (Bibliographic Retrieval Services) data bases, and ten data bases were identified as having a significant number of materials relating to all aspects of noise. (Note: the search strategy employed in the BRS system utilized free-text searching rather than controlled vocabulary, and although some loss of specificity of subject was demonstrated, a broader based coverage of concept was obtained.)

After identifying the ten data bases with a significantly high proportion of literature dealing with noise, the concept of noise was combined with various terms relevant to cardiovascular changes and/or disease states to produce a bibliographic listing of pertinent materials. Following, in alphabetical order, are the data bases accessed:

(1) AGRICOLA (CAIN) - 1975 to present; a cataloging and indexing data base of the National Agricultural Library, providing comprehensive, worldwide coverage of journals and monographs on agriculture and related subjects.

(2) BIOSIS (BIOL) - 1977 to present; a major English language service providing comprehensive worldwide coverage of research in the life sciences.

(3) DOE ENERGY (DOED) - 1974 to present; one of the world's largest sources of literature on energy and related topics. All manner of energy topics are included as well as related topics of environments, policy and conservation.

(4) GPO MONTHLY (GPOM) - 1975 to present; an on-line equivalent of Monthly Catalog of United States Publications. It includes all materials issued by all U.S. Federal government agencies including Senate and House hearings.

(5) INSPEC (INSP) - 1977 to present; the largest English language data base in the fields of physics, electro-technology, computers, and control.

(6) NATIONAL CLEARINGHOUSE FOR MENTAL HEALTH INFORMATION (NIMH) - 1969 to present; contains citations from books, journals, technical reports, workshop and conference proceedings in the area of mental health on a world-wide basis.

(7) NTIS (NTIS) - 1975 to present; consists of government sponsored research, development, and engineering, plus analyses prepared by Federal agencies, their contractors, or grantees.

(8) PSYCHOLOGICAL ABSTRACTS (PSYCH) - 1967 to present; covers the world's literature in psychology and related disciplines in the behavioral sciences.

(9) SMITHSONIAN SCIENTIFIC INFORMATION EXCHANGE (SSIE); a data base containing reports of both government and privately funded scientific research projects currently in progress or initiated and completed within the last two years.

(10) STATE PUBLICATIONS INDEX (IHSP) - 1976 to present; the single most comprehensive source of current state documents issued by the fifty states, Puerto Rico, and the Virgin Islands.

Two other data bases, not available on BRS, were considered to be potentially relevant. These data bases, EXCERPTA MEDICA and POLLUTION ABSTRACTS, were available and accessed through Dialog, the Information Retrieval Service of Lockheed. Following are brief descriptions of each:

> EXCERPTA MEDICA, 1974 to present, consists of abstracts and citations of articles from over 3,500 biomedical journals published throughout the world. It is the primary abstracting source for health sciences periodical literature published in Europe.
>
> POLLUTION ABSTRACTS, 1970 to present, is the leading resource for references to environmentally related literature on pollution, its sources, and its controls.

In addition to the computerized bibliographic searches, several manual searches were performed to identify older materials in indexes now on-line, and to identify materials possibly missed in indexes which do not yet have on-line access. These searches revealed a few classic studies, but in general, because of the quality of information sought, most of the literature acquired and the sources searched were limited to 1970 or later.

In addition to the traditional bibliographic methods, another major source utilized was extant bibliographies on the subject. Besides the bibliographies of relevant literature, the references found in the reports of Hattis and colleagues (1980), Welch (1979), and Taylor and others (1980) were compared to the citations identified by the various bibliographic searching techniques. Any relevant material not identified through the indexes, abstracts, or on-line data bases was acquired. These bibliographies proved quite useful in identifying unpublished sources.

2.2 Screening of Titles by Technical Team and Acquisition of Documents

From three major sources, On-Line Bibliographic Searching, Manual Bibliographic Searching, and Extant Subject Bibliographies, tentative bibliographies were compiled and sent to the team of experts. Potentially relevant documents were selected from these comprehensive bibliographies (comprised of approximately 1,300 citations) based on applicability to the project as a whole or any specific sub-specialty of the individual expert.

Attempts were made to acquire all documents requested by the team of reviewers. Of the approximately 800 articles, monographs, reports, etc. identified by the team as potentially applicable, the majority were available locally. Three hundred fifty-four were requested through interlibrary loan and a few documents, primarily monographs, were acquired through purchase.

Many of the documents on extra-auditory effects of noise were published in a Russian or one of the Eastern European journals. Primary access to these materials was made directly through the National Library of Medicine, although several requests were made to the British Lending Library.

One article, appearing in several bibliographies, was unobtainable because of an inability to verify its existence through standard bibliographic sources and the lack of a complete citation which would have enabled its location directly in the specific journal. Several citations were also unobtainable because of their status as unpublished reports with respective company policy governing their distribution.

In addition to interlibrary loan, the participating library purchased several items deemed essential to the study and not available through the extant document delivery network.

Following the acquisition and selection of highly relevant materials, bibliographies of those documents were screened for any potential documents or information souces not identified by the initial three search procedures. These items were then obtained for review. As a final step in the literature identification process, personal contact was made with recognized authorities in the field. This source proved invaluable in the identification of current research and unpublished reports.

2.3 Technical Translations

No attempt was made to obtain translations of current research in cardiovascular epidemiology in general. For the literature on the cardiovascular effects of noise exposure every attempt was made to acquire materials in translated format, or to obtain extant translations using sources such as Bibliography of Medical Translations, Technical Translations, and Translations Register Index. However, none of the foreign language articles, determined by abstracts to be specifically relevant, were found to be available in English translation. Translators available on a local basis after the majority of the foreign language materials had been obtained were those with a fluency in German, Japanese, and Rumanian. However, the greatest proportion of non-English language documents were translated by Duke Translation Service, associated with Duke University, and located in Durham, North Carolina.

Forty-seven articles were translated, the vast majority in Russian. Those translated by Duke were done through their interactive translation process, which provides for a final reading of the work by an expert in the field, prior to the typing of the final copy. This type of translation assures readability as well as comprehensibility.

Figure 2-1 gives a summary of the information processes used in the identification, acquisition and selection of relevant literature for review.

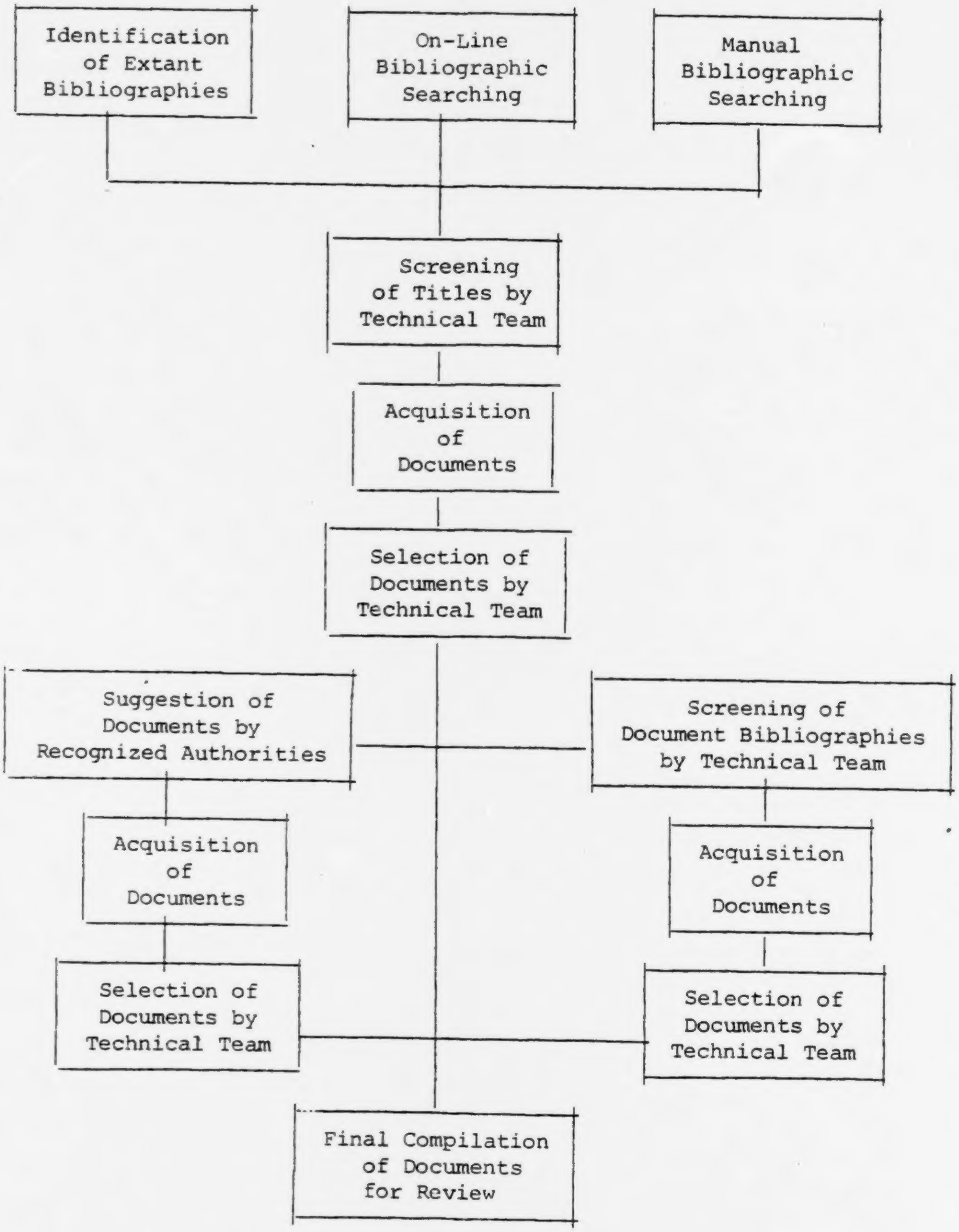
Identification of Extant Bibliographies
On-Line Bibliographic Searching
Manual Bibliographic Searching
Screening of Titles by Technical Team
Acquisition of Documents
Selection of Documents by Technical Team
Suggestion of Documents by Recognized Authorities
Screening of Document Bibliographies by Technical Team
Acquisition of Documents
Acquisition of Documents
Selection of Documents by Technical Team
Selection of Documents by Technical Team
Final Compilation of Documents for Review

Figure 2-1. Summary of Information Processes: Identification, Acquisition and Selection of Relevant Materials

Section 3

LITERATURE EVALUATION AND ANALYSIS

3.1 The Review Process

This critical analysis of the extant world literature was undertaken for the purpose of determining the extent to which studies reported to the scientific community are informative in judging the effects of noise on the cardiovascular system. It is based on thirty-six studies reported in the English language journals and forty-seven reported in the foreign literature and translated expressly for this review.

To insure an appropriate and scientifically sound analysis from the acoustical, medical/health effects and epidemiologic perspectives, a team approach was adopted. The team was composed of an audiologist, a cardiologist, a cardiovascular disease epidemiologist and the project director, also an epidemiologist. In addition, two local consultants (a medical epidemiologist and a cardiologist) and two external consultants (a researcher on noise and its potential health effects and a renowned cardiovascular epidemiologist) participated in the literature analysis process.

The review was accomplished as follows:

(1) A set of evaluative criteria was developed by the team, each member bringing to the task knowledge and methodologies appropriate to his discipline.

(2) Each relevant study was critiqued independently by each team member; the audiologist evaluated the research for the quality of the noise exposure component; the cardiologist focused on the assessment of the health effect studied; the epidemiologist rated each study for its adequacy in meeting methodologic criteria appropriate to epidemiologic design and analysis.

(3) Each expert reviewer assigned a numerical score to that component of the study which he evaluated, resulting in three scores for each study: a noise exposure rating, a health effects score and an epidemiologic-methodology score.

(4) An overall validity score was assigned each article by assuming that the lowest rating assigned to the three components evaluated represents

an appropriate overall rating because all major limitations have been taken into account.

(5) The project director summarized the research report, integrating the individual analyses of the team. These summaries are presented in Appendix B as annotated bibliographic material.

(6) The review team and consultants discussed the studies and summaries of the analyses and derived the major conclusions of the report.

The evaluative criteria and overall rating schemes are provided here as a framework for the presentation of the substantive findings from the literature analysis.

3.2 Evaluative Criteria

The assessment criteria addressed both substantive and methodologic aspects of the research and were derived from acoustic, medical and epidemiologic knowledge bases.

3.2.1 Noise Exposure

Five factors were considered in the evaluation of the noise parameter: noise description, instrumentation, environment in which measurements were taken, the measurement procedure used and quantity and quality of data about the subjects. Special attention was directed to assessing the nature and location of the noise source or sources, the relation of the individual to these sources, changes in the noise environment during the day, influence of background noise levels, noise exposure levels, the frequency of the noise, type of averaging method used, and subject information such as the adequacy of hearing threshold determinations. The five areas were judged on the amount of detail reported and the quantity of the data available. Table 3-1 outlines the criteria applied in judging the exposure variable, noise. Needless to say, lack of information on any criterion severely limits the value of a study for judging potential causal relationships between noise and cardiovascular health effects.

Table 3-1

Criteria for Judging the Exposure Variable - Noise

Terms used should be compatible with recognized standards such as ANSI S1.1-1960(R1976) American Standard Acoustical Terminology, ANSI S3.20-1973(R1978) American National Standard Psychoacoustical Terminology, S1.13-1971(R1976) American National Standard Methods for the Measurement of Sound Pressure Levels, and S1.4-1971(R1976) American National Standard Specification for Sound Level Meters.

I. Noise Description
   A. Type of noise (as: steady, nonsteady, impulsive, etc.)
   B. Frequency composition (as: pure tone, narrow-band, wide-band, specified)
   C. Levels: (in decibels) the type of noise must be indicated by the further modifier or context.
      "The physical quantity measured, the reference quantity, the instrument used, and the bandwidth or other weighting characteristic must be indicated."
      For air-borne sound, unless specified to the contrary, noise level is the weighted SPL called sound level; the weighting must be indicated. Sound Pressure Level (SPL) in decibels of a sound is 20 times the logarithm to the base 10 of the ratio of the pressure of this sound to the reference pressure. The reference pressure should be explicitly stated.
   D. Duration of exposure (in msec., sec., min., hrs., days, years, etc.)
   E. Source (as: machine, engine, musical instrument, etc.)

II. Instrumentation
   A. Type(s)
      Instruments used for noise measurement should be appropriate for the methods by which measurements are made, i.é., survey, field or laboratory methods as described in ANSI Standard S1.13-1971(R1976).
      - Sound Level Meter (SLM): "An instrument including a microphone, an amplifier, an output meter, and frequency weighting networks for the measurement of noise and sound levels in a specified manner."
        Type 1 (Precision) most precise instrument
        Type 2 (General Purpose)
        Type 3 (Survey) Least precise instrument
        Type S (Special Purpose)
        Note: Tolerances and specifications for SLM's are described in ANSI S1.4-1971(R1976).
      - Sound Analyzer: "A device for measuring the band-pressure level or pressure-spectrum level of a sound as a function of frequency."
      - Oscilloscope or Impulse Analyzer should be used to measure impulsive noise.
      - Dosimeter: device which accumulates total exposure to noise information as the wearer moves through environments having differing noise situations.
   B. Make & Model
      - Manufacturer plus Model name or number specified
      - Quality of the instrument relative to known techniques at the time of the study and relative to current knowledge

Table 3-1 (cont.)

C. Compliance with recognized standard such as ANSI or ISO Standards (cited in article)
   - All instruments used in the studies should meet or exceed recommendations of a recognized standard such as ANSI or ISO Standards.

III. Environment
   A. Type - Sound field, sound-treated room, open area, underwater, reverberant room, etc.
   B. Controlled or Uncontrolled - Conditions constant or conditions varying unpredictably.

IV. Measurement Procedure
   A. Type - Degree to which a standard technique is used. (Specified as to location of measuring device, etc.)
   B. Compliance with recognized standard such as ANSI or ISO Standards (cited in article).
      - All measurements should be accomplished in a manner compatible with guidelines published by ANSI, e.g., ANSI S1.2-1962(R1976) American Standard Method for the Physical Measurement of Sound and/or ANSI S1.13-1971(R1976) American National Standard Methods for the Measurement of Sound Pressure Levels.
        For example, reporting of measurement procedures should specify necessary information such as:
        - Calibration of instruments at least prior to and following measurements;
        - Correct manipulation of the instrument itself;
        - Correct and detailed recording of the measurements.

V. Subjects
   A. History of vocational noise exposure
      - Including all jobs which involved work around noise
      - History of previous and/or current involvement in a hearing conservation program
   B. History of avocational noise exposure
      - Including all non-working activities around noise
      - Nature and extent of any military experience
   C. Hearing thresholds obtained before and after noise exposure study and the adequacy of such determinations
      - With at least 14 hours away from the noise before testing is done
   D. History of ear disease
      - Where history of disease, problem identified and duration reported
   E. Otological examination
      - To insure that factors influencing hearing are identified (as impacted cerumen, m.e. disease, etc.)

VI. Investigative Personnel
   Although this information was not included in the scoring process, it is desirable.
   A. Professions - clearly stated versus not revealed by information available
   B. Qualifications - clearly stated versus not revealed by information available

3.2.2 Health Effects

A broad range of responses derived from the review of cardiovascular epidemiology were considered. These responses are shown in Table 3-2 and are described in Appendix C. Biochemical, physiological and pathological effects as well as clinical manifestations of cardiovascular disease were included because of their potential for enhancing adverse chronic states in response to noise. Because of the scope and complexity of these health effects, standard criteria for each, though not explicitly outlined on the assessment form, were determined and applied as appropriate. Table 3-3 shows the six factors considered in the assessment of the health parameter: diagnostic criteria, documentation of pre-existing cardiovascular disease, time relationships, natural course of disease states, risks of specific responses and methodology. Each criterion was judged according to both the quantity and quality of the detail provided in the research report.

3.2.3 Epidemiologic Methodology

Basically, epidemiology can be considered a two-stage sequence of reasoning: the first stage is the determination of a statistical association between an exposure variable such as noise and a disease or health state; the second stage is the derivation of causal inference from a pattern of observed associations. In this second stage of reasoning, at least four criteria are of equal importance to statistical significance in judging the causal nature of the association: (1) the strength and adequacy of the study design; (2) the strength of the association indicating the relative importance of possible etiologic factors; (3) the temporal relationship of the factors under study; and (4) the degree of exposure or dose-response relationship. The epidemiologic evaluation focused on these four criteria in judging the overall methodologic adequacy of the observational studies. Table 3-4 details the areas on which each study was evaluated.

3.3 Validity Ratings

Each study received three ratings, a noise exposure score, a health effects rating and an epidemiologic methodology score, derived in the

Table 3-2

Enumeration of Cardiovascular Responses to Noise

- I. Biochemical, Physiological and Pathological Effects
  - A. Biochemical
    - 1. Blood Lipids - Cholesterol, Triglycerides, Lipoproteins
    - 2. Coagulation - Platelet Function
    - 3. Adrenocortical Function - Corticosteroids
    - 4. Renin - Angiotensin Aldosterone System
    - 5. Prostaglandins
    - 6. Kallikrein - Kinin System
  - B. Physiological
    - 1. Cardiovascular
      - a. Blood Pressure
      - b. Cardiac Output - Myocardial Function
      - c. Peripheral Vascular Resistance
      - d. Peripheral Blood Flow
      - e. Heart Rate
      - f. Cardiac Work
      - g. Coronary Blood Flow - Myocardial Oxygen Consumption
      - h. Electrocardiographic
    - 2. Autonomic Function
      - a. Direct - Catecholamines
      - b. Indirect - Responses of Cardiovascular System
  - C. Pathophysiological (Pathological)
    - 1. Atherosclerosis
    - 2. Hypertension
    - 3. Arrhythmias
    - 4. Hypercoagulability
- II. Clinical Manifestations
  - A. Aggravation (Complicating Factors) - Acceleration of Underlying Disease States
  - B. Precipitation of Clinical Events in Existing Disease States
  - C. Development and Incidence of Disease States
    - 1. Atherosclerotic Diseases
      - a. Ischemic Heart Disease - Angina Pectoris
        - Myocardial Infarction
        - Sudden Death
      - b. Cerebrovascular Disease - Stroke
      - c. Peripheral Vascular Disease
    - 2. Hypertension and Hypertensive Cardiac Disease
    - 3. Other Clinical Manifestations or Complicating Factors of Cardiovascular Disease
      - a. Cardiac Arrhythmias
      - b. Congestive Heart Failure

Table 3-3

Criteria for Judging the Response Variable - Cardiovascular Health Effects

Disease states and risk factors were judged according to the following criteria.

I. Diagnostic criteria for clinical manifestations or diseases, i.e., New York Heart Association Classification.

II. Documentation of pre-existing cardiovascular disease.

III. Time relationships of exposure events to clinical manifestations, disease development, clinical events.

IV. The natural course of disease states.

V. Risk of specific clinical manifestations or pathophysiological responses.

VI. Methodology for determining response - variability of physiologic response measurements. This shall include but not be limited to procedures used, reliability and validity of the instrumentation, qualifications of personnel making determinations, consistency of the testing process.

Because of the scope and complexity of the biochemical, physiological, pathological, behavioral effects and clinical manifestations to be evaluated, standard criteria for specific health effects were determined and applied as appropriate.

Table 3-4

Criteria for Judging Epidemiologic Studies of the Nonauditory Effects of Noise on Man

I. Classification of Study Design
Statement of Objectives (for clarification, not to be rated)
- Stated in terms of anticipated risks (evidence of literature support)
- Clear and justified in terms of existing scientific information

A. Strength of the Design in Determining Etiology
1. Experimental Studies (Intervention)
   - Randomized Trial: of long-term effects
     : of short-term effects
2. Observational
   - Cohort or historical prospective: with individual exposure determined
     : "ecological" exposure as opposed to individual
     : hearing-loss exposure cohort
   - Case-control
   - Cross-sectional: prevalence
     : demographic studies of risk or mortality or morbidity using individuals classified on all variables
     : ecologic (classification based on aggregate measures as distinct from single individuals)
   - Case series with implicit controls (not acceptable but if there is reason to review, use appropriate criteria and justify)

B. Bias Potential in Design
The extent to which study design reported/controlled for bias such as exposure suspicion bias and recall bias.

C. Data Sources and Method of Data Collection
1. Primary versus secondary data sources
2. Data collected in a blind or double-blind manner - specify potential bias and reliability issues
3. Potential bias related to data source such as survival, self-selection identified. (Rate a specific bias potential only once in a given study.)

II. Sample

A. Type
- Random population sample (describe on characteristics such as age and sex)
- Retrospective cohort (exposure identified historically through records)
  : evidence of accounting for complete cohort; method of accounting for non-respondents justified
  : if not complete cohort by noise exposure, detailed description of method for selection with potential selection bias addressed
- Case-control Series
- Other samples from selected, but well defined populations

B. Sample size adequate for testing stated hypothesis (sample size calculations provided)

C. Follow-up
- Attrition - consider size of loss rate in each exposure category

D. Potential Bias and Its Control
1. Selection Bias - Criteria and rationale for inclusion and exclusion of subjects indicate potential bias was considered including healthy worker effect
2. Selective-Survival Bias
3. Migration Bias - Length of follow-up given with description of direction and magnitude of bias in results due to non-comparable follow-up

III. Specification of the Exposure and Response Variables

A. Noise Exposure as an Epidemiologic Variable
In addition to separately specified criteria:
- "Exposure" applied to individuals as opposed to "ecological" measure
- Completeness of exposure history - past to present
- Single versus multiple assessments of exposure for any given time period
- The extent to which the reliability and validity of exposure measurement is appropriate for assessing risk in large populations and/or selected groups at potentially high risk
- Potential measurement bias

B. Health Outcomes as Epidemiologic Variables
In addition to separately specified criteria:
- Extent to which reliability and validity of methods are appropriate for epidemiologic purposes
- Extent to which each individual was assessed on the response variable
- Extent to which the study depends on existing data from records: collected for study with reliability and validity checks built in versus dependent on well documented records versus collected for clinical or other than research purposes
- Potential measurement bias

Table 3-4 (cont.)

IV. Treatment of the Data: Analytic and Statistical Procedures for Judging Causal Significance
  A. The Strength of the Association
    1. Epidemiologic effect parameter used to quantify the strength of the association for categorical variables:
      - Relative risk
      - Attributable risk
      - Odds ratio
      - Population attributable risk (if appropriate)
    2. Epidemiologic effect measure used to quantify the association for continuous variables:
      - Correlation coefficient
      - Regression coefficient
    3. Temporal relationship
      Did study demonstrate that noise exposure preceded physiological response (important in long-term effects or chronic effects)?
    4. Consistency
      Were the observed relationships consistent with findings of other studies?
    5. Coherence
      Do study results conflict with generally known facts and biology of the disease?
  B. Appropriate statistical tests of significance and confidence intervals provided for risk assessments in 1 and 2 above
  C. When a meaningful relationship is observed, is a dose-response evident in the data?
    - Within the single study
    - In an ecological sense: Can data from several studies be evaluated together?
  D. Confounding
    Were potentially confounding variates (known strong risk factors) controlled through matching, stratification or statistical analysis?

following manner. Within each of these components, each categorical criterion set forth was scored "2" if met, "1" if partially met, and "0" if not met. An "I" was used to designate that an invalid criterion was applied, but the score "0" was assigned to that element. When a specific criterion was not applicable to a given study, it was so designated; that criterion was not included in the calculation of the numerical score. There were a possible 34 points for the noise exposure variable, 12 points for the health effects and 24 points for epidemiologic methodology. For comparison purposes, these numerical values were converted to a score ranging from 0-9 (lowest-highest) according to the percentage of criterion points met on the given component. See Appendix B for the assessment forms and conversion scale.

Next, studies were ranked as to overall validity. It was assumed that the lowest of the three scores assigned by the experts on noise, health and methodology represents a fair score of overall validity. This rating implies that valid measures of the exposure and outcome variables as well as adequate methodology is required, and that in the absence of any one of these components, the study becomes less than fully informative for assessing the effects of noise on the cardiovascular system.

3.4 Introduction to Critical Analysis of the Literature

The impact of environmental noise on human health has been considered by many investigators, but there is little consensus as to the nature or extent of the potential adverse effects of noise on the cardiovascular system. This section of the report summarizes and evaluates the epidemiologic evidence for and against the hypothesis that long-term exposures to noise adversely affect cardiovascular response in man.

For analysis purposes, the papers were categorized as containing adverse, some adverse, or no adverse effects according to the conclusions stated by the author(s). A study listed under "adverse effects" reports deviations from normal which the author infers to be detrimental to health. A study categorized as "some adverse effects" presents several findings, at least one of which the author infers to be detrimental to health. This category includes reports showing differential effects among population subgroups and/or noise effects for one health outcome, but not another. It is important to note that this is a grouping for convenience of reporting. There is no a priori reason why all subgroups should be equally sensitive to the same noise exposure and all show the same effect. Likewise, there is no a priori reason to expect noise to produce a similar influence on all aspects of the cardiovascular system. In fact, diverse susceptibilities of populations to disease and individual variability in response to environmental stimuli would suggest the potential for differential effects.

The articles were further classified by the setting of the study, in general reflecting the source of environmental noise. These groupings were (1) industrial noise; (2) transportation noise (airport, road, railroad), neighborhood noise and noise exposure determined from general community surveys; and (3) laboratory experiments.

For convenience, critiques of the literature originally published in English are presented, followed by critiques of the translated literature. In Appendix A, Tables A-1 through A-10 give a concise summary of each paper with the author's conclusions. For a more detailed critique of each study, see Appendix B.

The relatively poor quality of the identified papers is reflected in the individual component and overall ratings of the reviewers as shown in Table 3-5. The proportions of studies meeting more than fifty percent of the evaluative criteria were as follows: on the noise component, 6% of the English studies and 11% of the translated research; on the health outcome component, 33% of the English and 32% of the translated studies; and on the epidemiologic methodology component, 42% of the English and 11% of the translated studies. When the lowest of the three component scores is taken as the overall validity score, no study reported in the English literature and only one in the translated literature was rated higher than "4" on the 0-9 scale (see Tables 3-6 and 3-7). These ratings indicate that the literature is less than fully informative for the task of judging the association between noise and cardiovascular effects.

Table 3-5

Summary of English and Translated Literature by Percentage of Evaluative Criteria Met

Scale Value	Percent of Criteria Met	Noise		Health		Methodology		Overall Score	
		English	Translated	English	Translated	English	Translated	English	Translated
9	91-100			5.5(2)	4.3(2)				
8	81- 90			16.7(6)	2.1(1)	2.8(1)			
7	71- 80		2.1(1)	2.8(1)	10.6(5)	13.9(5)			
6	61- 70	2.8(1)	4.3(2)	5.5(2)	12.8(6)	11.1(4)	4.3(2)		
5	51- 60	2.8(1)	4.3(2)	2.8(1)	2.1(1)	13.9(5)	6.4(3)	0.0	2.1(1)
4	41-50	22.2(8)	6.4(3)	8.3(3)	17.0(8)	16.9(6)	17.0(8)	16.7(6)	4.3(2)
3	31-40	16.7(6)	14.9(7)	25.0(9)	8.5(4)	25.0(9)	29.8(14)	16.7(6)	12.8(6)
2	21-30	27.8(10)	31.9(15)	16.7(6)	14.9(7)	8.3(3)	25.5(12)	19.4(7)	21.3(10)
1	11-20	22.2(8)	21.3(10)	- (0)	21.3(10)	5.6(2)	8.5(4)	25.0(9)	34.0(16)
0	0-10	5.6(2)	14.9(7)	16.7(6)	6.4(3)	2.8(1)	8.5(1)	22.2(8)	25.5(12)
	Totals	100.1(36)	100.1(47)	100.0(36)	100.0(47)	100.1(36)	100.1(47)	100.0(36)	100.0(47)

Table 3-6

Studies Ranked by Overall Score from Highest to Lowest Shown with Ratings on Noise Exposure, Health Effects and Methodology English Literature

	Noise	Health	Method	Overall Ranking Score
Brown et al (1975)	4	8	6	4
Cohen et al (1980a)	4	8	6	
Cohen et al (1980b)	4	9	6	
Cohen et al (1981c)	4	9	7	
Dega et al (1977)	5	4	4	
Raytheon (1975)	4	6	5	
di Cantogno et al (1976)	6	8	3	3
Jonsson et al (1977)	3	3	4	
Knipschild (1977a)	3	3	4	
Mosskov et al, II (1977a)	4	3	3	
Mosskov et al, III (1977b)	4	3	3	
Mosskov et al, IV (1977c)	4	3	7	
Andriukin (1961)	2	3	3	2
Cuesdean et al (1977)	2	2	4	
Malchaire et al (1979)	2	5	7	
Manninen et al (1979)	2	4	5	
Ohrstrom et al (1979)	2	2	2	
Semczuk et al (1971)	3	2	3	
Yazburskis (1971)	2	2	2	
Drettner et al (1975)	1	7	4	1
Friedlander et al (Undated)	1	6	5	
Gibbons et al (1975)	1	2	3	
Kavoussi (1973)	1	3	2	
Knipschild (1979c)	1	2	7	
Lees et al (1979a)	1	8	8	
Lees et al (1980b)	1	3	5	
Parvizpoor (1976)	1	8	6	
Proniewska et al (1972)	3	4	1	
Antonova (1971)	2	0	0	0
Frerichs et al (1980)	2	0	7	
Graeven (1974)	2	0	3	
Hannunkari et al (1978)	3	0	5	
Hedstrand et al (1977)	0	3	3	
Knipschild (1977b)	3	0	3	
Meecham et al (1979)	2	0	1	
Takala et al (1977)	0	8	4	

Table 3-7
Studies Ranked by Overall Score from Highest to Lowest
Shown with Ratings on Noise Exposure, Health Effects and Methodology
Translated Literature

	Noise	Health	Method	Overall Ranking Score
Quaas et al (1970)	6	9	5	5
Burger et al (1975)	4	9	4	4
Ising et al (1979)	6	4	6	
Folprechtova-Stenzlova et al (1966)	5	7	3	3
Klotzbuecher (1976)	3	4	5	
Mariniako et al (1975)	3	6	3	
Sanova (1975)	4	3	3	
Stasiow et al (1974)	4	7	3	
Suvorov et al (1979)	3	5	5	
Britanov (1979)	5	3	2	2
Demeter et al (1979)	2	2	2	
Geller et al (1963)	2	4	2	
Graff et al (1968)	2	4	4	
Jansen (1959a)	2	2	3	
Kachnyi (1977)	2	6	2	
Kalicinski et al (1975)	2	4	3	
Pilawska et al (1977)	7	2	4	
Rumiantsev et al (1971)	2	7	2	
Shatalov et al (1962a)	2	3	3	
Andrukovich (1965)	2	6	1	1
Cieslewicz (1971)	1	7	3	
Gel'tishcheva (1980)	2	4	1	
Jansen (1961b)	3	1	2	
Jirkova et al (1965)	2	1	3	
Kangelari (1966)	2	1	2	
Khomulo et al (1967)	1	7	2	
Kobets et al (1972)	2	1	3	
Lanzetta et al (1979)	1	8	4	
Pokrovskii (1966)	1	2	3	
Shatalov (1965b)	1	3	2	
Shatalov et al (1969c)	1	2	2	
Terentiev et al (1969)	2	1	2	
Troianskii et al (1971)	1	6	4	
Von Eiff et al (1980)	1	6	6	
Zvereva et al (1975b)	3	1	1	
Barhad et al (1969)	1	0	3	0
Capellini et al (1974)	0	6	1	
Grusha (1974)	0	1	0	
Kanevskaia et al (1977)	2	0	3	
Koszarny et al (1976)	0	1	2	
Liubashevskaia et al (1976)	2	0	2	
Meinhart et al (1979)	0	2	4	
Paranko et al (1974)	1	4	0	
Shatalov et al (1970d)	0	1	4	
Tavtin (1976)	3	0	3	
Vopilkina (1959)	0	4	0	
Zvereva et al (1975a)	3	1	0	

3.4.1 Noise-Induced Hearing Loss as a Surrogate for Noise Exposure

Nine studies presented in this review of the literature used noise-induced hearing loss to indicate long-term noise exposures when the hearing-impaired subjects were not actually known to have a history of exposure to specific noise levels. The use of noise-induced hearing loss as a surrogate measure for noise exposure may result in misclassification, that is, a subject may be placed in the wrong "exposure" category. Such misclassification could affect the direction and strength of the association between noise exposure and blood pressure. There has been and continues to be considerable debate about the interpretation of noise-induced hearing loss and its relationship to specific noise exposures (Glorig, 1980). Therefore, the findings from studies employing surrogate measures must be interpreted with caution. The nine studies are discussed in this review according to the conclusions derived by the authors (Drettner et al, 1975; Jonsson et al, 1977; Takala et al, 1977; Hedstrand et al, 1977; Demeter et al, 1979; Lees et al, 1979; Manninen et al, 1979; Meinhart et al, 1979; A. Cohen et al, 1980a).

Hearing loss considered to be noise induced has been used as a surrogate measure of noise exposure based on the following assumptions: (1) the first noticeable effect of exposure to loud sound is the increase of the hearing threshold, experienced after termination of noise exposure; (2) hearing levels decrease gradually and if the noise is not too loud and exposure is not too long, the hearing threshold will return to normal in a few hours or days (temporary threshold shift); if exposure continues, a residual hearing loss (permanent threshold shift) will occur; (3) development of noise-induced hearing loss is a gradual process, normally progressing over a period of years, affecting high frequency hearing acuity first and eventually spreading toward lower frequency impairment; (4) noise-induced loss is characterized by declining sensitivity to high frequencies with the loss appearing first and more severe for the 4000 and 6000 Hz frequencies; (5) noise-induced hearing loss is a sensorineural loss which will stabilize within approximately two weeks after the last exposure; (6) generally equal magnitude of impairment occurs in each ear and interaural differences greater than 20-25 decibels should be regarded with suspicion unless there is an unusual exposure condition affecting only one ear; (7) as people age their hearing worsens (presbycusis). Presbycusis is sometimes taken into account when the degree of noise-induced hearing loss is determined (Glorig, 1980 and Moller, 1977).

The major problem in accepting noise-induced hearing loss as a surrogate measure for noise exposure is the unknown relationship between hearing impairment and blood pressure. Welch (1979, p.7) noting the hypotensive trend among the hearing impaired suggested several hypotheses: "An alternative explanation would be that noise tends to increase the prevalence of hypertension among those who can hear it; that extreme hearing impairment itself has a protective effect against blood pressure elevation by shutting the noise out; and that, once established, deafness may even favor the regression of a previously established hypertension trend." As Welch goes on to point out, the observed results may also reflect chance aberration. Hattis and Richardson (1980, p.120) also share some of Welch's concern about the use of hearing impairment as a proxy measure for noise exposure. They cited a study by Borg and Moller who observed that the Okamoto strain of rats suffered markedly worse hearing impairment in response to noise than their genetically normotensive counterparts. Blood pressures in worker groups with different degrees of hearing impairment must be interpreted with caution when inferring causal relationships to noise exposure.

3.5 Critical Review of the English Literature

3.5.1 Adverse Effects: Blood Pressure as the Major Response Variable

Five researchers reported adverse blood pressure effects from investigations of long-term industrial noise exposure and one group of investigators recently reported adverse blood pressure effects among school children exposed to aircraft noise. Ratings in Table 3-8 demonstrate that the identified studies in which the authors report adverse effects are of variable quality. Specific findings are described below.

Table 3-8
Studies Ranked by Overall Score, Categorized by Exposure Setting and Nature of Findings* as Reported by Author(s)
English Literature

	Scores			
	Noise Exposure	Health Effects	Methodology	Overall Score
I. Industrial Noise - Adverse Effects Reported by Author				
Dega et al (1977)	5	4	4	4
Raytheon (1975)	4	6	5	4
Jonsson et al (1977)	3	3	4	3
Andriukin (1961)	2	3	3	2
Manninen et al (1979)	2	4	5	2
Ohrstrom et al (1978)	2	2	2	2
Semczuk et al (1971)	3	2	3	2
Friedlander et al (Undated)	1	6	5	1
Gibbons et al (1975)	1	2	3	1
Parvizpoor (1976)	1	8	6	1
II. Industrial Noise - Some Adverse Effects Reported by Author				
Mosskov et al, III (1977b)	4	3	3	3
Cuesdean et al (1977)	2	2	4	2
Yazburskis (1971)	2	2	2	2
Kavoussi (1973)	1	3	2	1
Proniewska et al (1972)	3	4	1	1
Antonova (1971)	2	0	0	0
Hannunkari et al (1978)	3	0	5	0
III. Industrial Noise - No Adverse Effects Reported by Author				
Brown et al (1975)	4	8	6	4
A. Cohen et al (1980a)	4	8	6	4
Malchaire et al (1979)	2	5	7	2
Lees et al (1980b)	1	3	5	1
Lees et al (1979a)	1	8	8	1
IV. Transportation Noise, Neighborhood Noise and Community Noise - Adverse Effects Reported by Author				
S. Cohen et al (1980b)	4	9	6	4
Knipschild (1977a)	3	3	4	3
Knipschild (1977b)	3	0	3	0
Meecham et al (1979)	2	0	1	0
V. Transportation Noise, Neighborhood and Community Noise - Some Adverse Effects Reported by Author				
S. Cohen et al (1981c)	4	9	7	4
di Cantogno et al (1976)	6	8	3	3
Mosskov et al, II (1977a)	4	3	3	3
Mosskov et al, IV (1977c)	4	3	7	3
Graeven (1974)	2	0	3	0
VI. Transportation Noise, Neighborhood and Community Noise - No Adverse Effects Reported by Author				
Drettner et al (1975)	1	7	4	1
Knipschild (1979c)	1	2	7	1
Frerichs et al (1980)	2	0	7	0
Hedstrand et al (1977)	0	3	3	0
Takala et al (1977)	0	8	4	0

*A grouping for convenience of reporting.

Adverse effects: the study shows deviations from normal which the author infers to be detrimental to health.

Some adverse effects: the study presents several findings, at least one of which the author infers to be detrimental to health. This category includes studies showing differential effects among population subgroups and/or noise effects for one health outcome but not another.

3.5.1.1 Industrial Noise and Blood Pressure

As early as 1961, Andriukin reported that hypertension was found more frequently in workshops with intense noise (sorting room - 103 db*, automatic lathes - 103 db, and ballbearing workshops - 120 db) than in the less noisy tool-making workshop (93 db). He further reported that morbidity increased with the duration of employment, particularly after five years work in the noisiest workshop, but he failed to provide supporting data. Although the author gives little information as to subject characteristics and the noise stimulus, it was noted that approximately fifty percent of the workers had been employed in the plant for more than ten years and in work that had changed little. Blood pressures of 676 men and 556 women were recorded during work, after a ten minute break and repeated until constant values were obtained. Blood pressures of 130/90 mm Hg were taken as the upper limit of normal for subjects under 40 years of age and 140/90 mm Hg for subjects over 40 years of age. The author failed to state whether high blood pressure was defined by either diastolic or systolic readings or whether both systolic blood pressure and diastolic blood pressure have to fall into the upper limits of normal for classification as a hypertensive. Since blood pressure increases with age, systolic blood pressure more so than diastolic blood pressure, classification by either systolic blood pressure or diastolic blood pressure may reflect aging in the group rather than noise exposure. Thus, statistically controlling for age effects becomes important. The prevalence data are presented by ten year age groups, but there is no evidence in the analysis of statistically controlling for age, sex, socio-economic class, medications, co-morbidities and rotating shifts. No gradient in hypertension prevalence as noise level increased in the workshops within the age groupings was noted as might be expected if noise was responsible for the adverse effects and selection bias was not operating. Andriukin treated the apparent cross-sectional observations as incidence data. Although it is possible, it is not very probable, that true exposure cohorts of workers within the four worksites were selected for study.

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* When no weighting network is specified by the author, it is not stated in this report. In most cases "A" weighting is understood.

In the 1970's, other groups of investigators employing cross-sectional designs reported adverse blood pressure effects. Among these, Parvizpoor (1976) concluded from his work that men employed at textile mills have a significantly greater risk of developing hypertension than other workers and that this difference appeared at relatively young ages (30-39) and increased with length of employment. He based his conclusions on a cross-sectional survey of 812 male weavers from three textile mills with noise levels of 96 dBA and 412 randomly selected controls of similar socio-economic status without occupational noise exposure. Blood pressure readings were classified according to the World Health Organization criteria as hypertensive when systolic blood pressure of 160 mm Hg or more or diastolic blood pressure of 95 mm Hg or more was observed. Borderline hypertension was defined as blood pressure between the hypertensive values and 140 mm Hg systolic or 90 mm Hg diastolic. Although the study is plagued with methodological problems and potential measurement error, Parvizpoor offered some evidence in favor of a noise and blood pressure relationship after age was controlled by stratification. He failed to state the strength of the relationship quantitatively, but from the prevalence data available, a derived crude odds ratio of 4.1 (odds of developing hypertension given high noise exposure) suggests a strong association. If one can accept length of employment as an indicator of noise exposure duration, the data showed an increase in prevalence of hypertension with an increase in length of employment. However in this latter analysis, age was not taken into account. A major weakness of this study in the textile mills of Iran is the lack of specification of the noise exposure parameter.

An unpublished student project utilizing the medical records of civilian employees of the Curtis Bay Shipyard concluded that the relative risk for developing hypertension was greater in those exposed to prolonged loud noise than in comparable age-race controls (Friedlander et al, undated). The authors described their study as historical prospective in design, but provided no evidence of identification of a specific cohort with appropriate follow-up. It is unlikely that the medical records represented a complete cohort of workers. Four hundred forty-one males were selected after many exclusions such as employment of less than five years, hypertension diagnosed on first clinic visit, inadequate blood pressure measurement or unsuitable work history, race not available. The records were subdivided according to noise exposure levels: office workers exposed to low noise of less than 70 dB; skilled workers employed in shops having moderate noise levels of about 70-79 dB and skilled workers intermittently exposed to noise greater than 80 dB. The latter two groups were combined because of the small sample size. Elevated systolic blood pressure was defined as greater than 140 mm Hg and elevated diastolic pressure as greater than 90 mm Hg. Data were stratified on race and age, but apparently there was no statistical controlling for potential confounders such as obesity, social class, physical exertion, other diseases, etc. Relative risks were reported for each race-age group with statistically significant differences observed only for 35-44 year olds. For the high noise group, the risk of developing elevated systolic blood pressure was 6.4 times that of the low noise group and of developing elevated diastolic pressure, 2.8 times that of the low noise exposed workers. While the reported relative risks are impressive, the results must be questioned because of the severe methodological problems in the study.

Jonsson and Hansson (1977) in Sweden and Manninen and Aro (1979) in Finland studied the effects of prolonged noise exposure on rise in blood pressure utilizing hearing loss levels of individual workers as surrogate measures for noise exposure. A significantly larger proportion of persons with hypertension was found by Jonsson and Hansson in the noise-induced hearing loss group compared to the normal hearing group. Noise-induced hearing loss was defined as a loss of acuity amounting to 65 dB or more at either 3000, 4000 or 6000 Hz. Mean systolic blood pressure values for subjects with impaired hearing was 145.2 compared to 132.6 among controls; mean diastolic blood pressures were 88.6 compared to 80.6. The authors offered the explanation that prolonged exposure to a stressful stimulus may have caused repeated rises in blood pressure leading to circulatory adaptations and a permanent rise in blood pressure.

Manninen and Aro's findings from a cross-sectional sample of workers in engineering factories classified by hearing loss were partially supportive of Jonsson and Hansson's observations. Their data demonstrated that exposure to noise first elevates the systolic blood pressure and to some extent the diastolic blood pressure, but that at severe hearing loss levels (continuing exposure) the systolic blood pressure tends to return to normal with the diastolic blood pressure either rising or falling. It should be noted, however, that these interpretations were derived from cross-sectional, not prospective, data. In neither of the latter two studies were noise levels reported in association with hearing loss levels.

3.5.1.2 Transportation, Neighborhood and Community Noise and Blood Pressure Response

One of the better studies, that of S. Cohen et al (1980b), assessing effects of aircraft noise on children, demonstrated findings supportive of a positive association between noise exposure and blood pressure levels. In this 1977 community survey, all children without hearing impairment and in the third and fourth grades of the four noisiest schools located in the air corridor of the Los Angeles International Airport were group-matched on ethnic, racial and socio-economic level to an equal number of classes selected from three quiet schools. Sound levels, without the children present, were measured inside each classroom with Tracoustics Sound Level Meters for a one hour period in the morning and a one hour period in the afternoon. The overall mean peak sound level in terms of dB(A) for classrooms in noise-schools was 74 dB with the highest reading at 95 dB. For classrooms in quiet schools, mean peak sound level and highest readings were 56 dB and 68 dB respectively.

An automatic blood pressure recorder was used to take the readings for 142 children from noise-schools and 120 from quiet-schools. Children were prepared for the procedure, the mean of the second and third blood pressure readings taken on consecutive days was used and coders were unaware of the noise conditions. Ethnicity, social class, race, ponderosity and mobility but not age, were controlled in the analysis. Blood pressure was observed to increase with years of exposure (defined as years enrolled in school), but the age effect needs to be explored since the increase was greater in the quiet-school children than in the noise-school subjects.

In a follow-up study, Cohen and colleagues (1981c) looked at changes in the blood pressure response of children who were moved from noisy classrooms in 1977 to noise-abated classrooms in contrast to changes in blood pressure of children exposed both years in noise-impacted rooms. At the time of the follow-up study in 1978, the Digital Acoustics (DA 605), B and K (4426), and General Radio (1945) noise level analyzers were available and used because of their increased sensitivity, accuracy and ability to provide various measures of noise over time. Sound levels, without children, were measured inside each classroom for one hour during the morning and one hour during the afternoon. Microphones were placed approximately three feet from the ground in the center of the room. Noise level was averaged on an energy basis over each hour period. A reanalysis of data from 1977 with classrooms categorized as noisy, abated and quiet showed mean peak noise levels of 79.06 dB for noisy classrooms, 63.17 dB for abated classrooms and 56.60 dB for quiet classrooms. In 1978, the mean peak noise level in noisy classrooms was 91.50 dB(A) and in the abated classrooms was 71.27 dB(A). Cohen and colleagues (1981c) report that the mean peak dB(A) measures in 1978 were higher than those recorded in 1977 because more sensitive automated equipment was used in 1978.

Although on reanalysis the cross-sectional data showed higher blood pressures for noise-school children than for quiet-school children, no significant differences in blood pressure were observed for the 39 noise-exposed children who had experienced a year in a noise-abated classroom compared to the continuously noise-exposed children. Unfortunately, this well designed survey suffered from a major problem which frequently plagues longitudinal research. A relatively high proportion of the noisy-school children with high blood pressure were lost to attrition. Thus, the data from the longitudinal and more powerful design is of little value in judging a causal relationship between blood pressure and noise.

3.5.2 Adverse Effects: Parameters Other than Blood Pressure as Major Response Variables

3.5.2.1 Industrial Noise and Health Parameters Other than Blood Pressure

Other investigators have found associations between high noise levels and diagnosed medical problems, CRIS index of cardiorespiratory efficiency, fatigue and other general symptoms and 17-ketosteroids. One such study was conducted by the U.S. Raytheon Service Company among workers in a plant producing large pressure boilers. The investigators compared record entries in the worker medical files for two-year periods just before (1969-70) and after the start of a hearing conservation program (1972-73). In high-noise areas comparisons in health status were made before and after the introduction of hearing conservation measures with consideration taken of degree of compliance. Workers in the low noise areas were studied for the same time periods to identify changes in health due to other environmental modifications and to detect any differences in the prevalence of extra-auditory problems between the high noise/hearing protected group (434 workers) and low noise group (432 workers) as a result of the conservation program. For all medical problems, a difference was demonstrated between the years prior to (1969-70) and after the implementation of the hearing conservation program (1972-73). Workers judged to have always used their ear protectors showed the greatest reduction in problems while those rated as never using the hearing protectors experienced the smallest relative change in health problems. It is not surprising that these differences failed to appear in the cause-specific data since the sample size was small and few new cases of cardiovascular disease could be expected to develop within the study period.

This study, considered to be one of the better among the research reported in the English literature, was based on the premise that a reduction in disease with the reduction of noise exposure through the use of hearing protectors, would be indicative of a causal relationship between high noise and the incidence of medical problems. It was somewhat of a natural experiment which approximated a non-equivalent control group design as described by Campbell and Stanley (1963). Several methodological limitations of the Raytheon study were that the groups were not naturally assembled collectives and the experimental variable was not totally under the investigator's control and randomly assigned. For ethical, practical and regulatory reasons, all workers exposed to high noise were offered hearing protectors with no random assignment into the groups. Extent of the use of the ear protectors was poorly documented. Extant medical records were employed to measure health outcomes. Coders were unaware of the exposure conditions. However, quality of the data was poor due to the inclusion of "diagnosed medical conditions" based solely on the verbal reports of the worker. Eight diagnostic categories other than the cardiovascular diseases of interest were reported. Diagnosed cardiovascular disease, especially by type such as acute myocardial infarction, would be a strong measure of epidemiological consequence. However, in this study, the results for cardiovascular disease are not surprising since noise exposure would have to be an extremely potent risk factor for differences in overt disease to be detected in such small samples over a relatively short period of time.

In a mixed cross-sectional/experimental design, Semczuk and Gorny (1971) at the Medical Academy in Lublin, observed 100 men at their workstands where the intensity of noise was 80-115 dB and 50 individuals who worked in noise not exceeding 65 dB. They reported that in 73 percent of the examined men, after eight hours of work in high noise, the CRIS value decreased by 2-8, while such changes were not observed in the control group. A decrease in CRIS, a measure of vital capacity, apnea and heart rate, was used to imply decreased cardiorespiratory efficiency. These researchers had derived similar conclusions from an experiment during which 50 persons were tested during stimulation of the auditory organs. Contrary to the authors' interpretation, findings are judged to provide little or no support to the hypothesis that noise produces adverse cardiovascular effects because the outcome measure, CRIS, is of unknown clinical significance.

Ohrstrom and Bjorkman (1978) demonstrated an increase in fatigue with high noise levels and long exposure. The authors implied that fatigue may be related to heart rate and blood pressure, but no objective data were provided. Although noise levels were measured in the machine and textile industries studied, the outcome data were very subjective and the analysis was lacking in control of potential confounders such as age and sex. In fact the investigators emphasized that the material was derived from two different populations - older males at demanding work for long periods of employment compared to younger females at monotonous work for short periods of employment.

3.5.2.2 Transportation, Neighborhood and Community Noise and Health Parameters Other than Blood Pressure

The work of Knipshcild (1977a and b), although based on cross-sectional, ecological data, lends some support to the proposition that noise adversely affects the cardiovascular system. In a survey of general practice contact rates in a community near Schipohl Airport, Amsterdam (1977b), he found an apparent gradient of increasing contact rates for cardiovascular disease from the low to high noise areas. Data from a cross-sectional community survey (Knipschild, 1977a) of the same area were also suggestive of a dose-response relationship: the percentage of participants with hypertension increased with increase in aircraft noise measured in the center of each village studied. A widely used descriptor, Noise and Number Index (NNI), was used to assess aircraft noise within the living areas surrounding the airport. Both surveys are subject to potential misclassification error since the actual exposure of individual subjects was unknown and the actual noise levels over the six year period of presumed exposure were estimates of widely varying frequencies of the noise.

The community survey sample of 2233 in the high noise area and 3595 in the low noise area represented only 43 percent of those invited to participate. Hypertension was defined as systolic blood pressure greater than 175 mm Hg and/or diastolic pressure greater than 100 mm Hg and was obtained from screening survey data. Other measures of cardiovascular response were diagnosis of angina pectoris, pathological electrocardiogram and heart shape and the taking of cardiovascular drugs. Knipschild reported that age and sex were controlled in the analysis; smoking, obesity and social class differences were observed, but the extent of controlling for these variables in the analysis is not clear. Applying a regression analysis, he concluded that the prevalence of cardiovascular disease appeared to increase with increase in noise levels. In summary, the limitations of this community survey are the low response rate, possibly due to participation costs, and incomplete controlling of potentially strong confounders such as social class and smoking. In addition there is the possibility of inferring an association between noise and cardiovascular disease from area measures of noise exposure when the association would not be observed if the noise exposures of individual participants were related directly to their health states. In his study of contact rates, Knipschild took age and sex into account in the analysis, but failed to control for socio-economic status and provided no information as to the proportion of noise complaints relative to all other disorders.

The findings reported by Meecham and Shaw (1979) of higher death rates for stroke and cirrhosis of the liver due to noise exposure from the Los Angeles airport have been refuted by Frerichs and colleagues (1980). Unlike the earlier study, Frerichs et al compared age-race-sex-cause specific death occurrences in the noise exposed and control areas and found no appreciable differences. Although it is possible that no associations were observed due to errors in diagnosis and reporting of deaths and the ecological nature of the data, the Vital Records mortality study of Frerichs et al (1980) is much more methodologically sound than that of Meecham and Shaw (1979).

3.5.3 Some Adverse Effects: Blood Pressure as the Major Response Variable

3.5.3.1 Industrial Noise and Blood Pressure

One study, weak in methodological rigor, reported an increase in hypertension among workers over 40 years of age exposed to silo noise, but no increase in blood pressure among younger noise-exposed workers (Kavoussi, 1973). Hypertension was defined as 140/90 or greater and was classified using the average of three blood pressure readings. The author's conclusions are questionable because the study failed to meet several evaluative criteria. It lacked a nonexposed control group, assumed length of employment measured duration of exposure, was cross-sectional and assessed only 66 percent of the employees, provided no noise measurements, applied no inferential statistics, and except for age stratification made no attempt to control for potentially confounding variables.

3.5.4 Some Adverse Effects: Parameters Other than Blood Pressure as the Major Response Variable

3.5.4.1 Industrial Noise and Health Parameters Other than Blood Pressure

An investigation of locomotive engineers by Hannunkari (1978) applied the most rigorous design of the studies indicating some adverse cardiovascular effects. It included a historical prospective analysis of mortality data for engineers with clerks and trainmen as reference cohorts as well as cross-sectional data on symptoms, complaints and disabilities. The mortality experience of some 437 engineers employed on December 1, 1955, and followed througn December 31, 1963, was compared to that of every second trainman (N=1575) and to all railroad clerks (N=1224) employed on December 1, 1955.

Although other results were difficult to interpret, engineers in all five age groups were observed to have the highest mortality due to diseases of the circulatory system. Unfortunately, the study suffers from several major shortcomings. The exposure variable of interest, noise, was poorly described although apparently assessed at some time in a hygienic survey of locomotive cabs. The noise exposure, reported as variable with 45 percent of the measured equivalent noise levels exceeding 85 dB(A) during a 0.5-2 hour measuring period, was clearly confounded by unassessed vibration levels. Information as to the sources, completeness and verification of the mortality and disability data was lacking. The investigators were rather vague as to their use of inferential statistics and apparently made no attempt to statistically control for age, comorbidities, drug use, or exercise in their analysis of morbidity. Audiometric examinations at initial employment and periodic intervals, reportedly showing 17 percent hearing loss among engineers, were not analyzed further.

Cuesdean and colleagues (1977) studied cardiovascular and hearing disorders among operatives in a Rumanian rubber plant. Noise levels from a variety of sources, ranging from 85-106 db were reported for 160 subjects, not exposed to toxic substances. Mean duration of exposure was 6 years in the 21-40 year old group and 10 years in the older workers (41-60 years). A group of 160 men and women similarly engaged in light manual labor served as controls. Subjects were also assessed as to hearing thresholds, smoking history, obesity and excess of animal fat in the diet, all of which may be potential confounding factors in the relationship between noise and cardiovascular disease. The state of the cardiovascular system was evaluated by three measures: hypertension, probably defined as greater than or equal to 140/90, electrocardiographic alterations and evidence of neurocirculatory asthenia. The authors reported hypertension among 8.1% of the operatives exposed to noise and hearing loss among 16.1%. They concluded that electrocardiographic alterations at indices 3-1, 3-3 and 9-2 according to the Minnesota code, were more frequent among men working in permanently intense noise than among others. The highest incidence was that of upward deflection of segment ST. Several problems emerge in the evaluation of this research. Firstly, even though the electrocardiographic changes are significant statistically, it is not clear that they are of any clinical significance relative to disease onset as suggested by the investigators. Secondly, the study design, sample selection, noise parameters and health measures were not described well, nor in detail. Thirdly, no multivariate analyses were performed capitalizing on the data available relative to sex, smoking, weight and diet.

3.5.4.2 Transportation, Neighborhood and Community Noise and Health Parameters Other than Blood Pressure

One study of general noise exposure reported some adverse effects of noise, but the weak study design limits its usefulness. A study in Hayward, California, determined degree of exposure to aircraft noise with the Noise Exposure Forecast measure and health status with a symptom checklist (Graeven, 1974). Graeven concluded that airplane noise was the third most important factor in determining health problems. Noise awareness and annoyance reactions were more important than the noise level. This conclusion was based on a 20 percent quota of females from four exposed areas and a control city, with unreported response rates, and varying age distributions within the areas which were not controlled in the analysis.

3.5.5 No Adverse Effects: Blood Pressure as the Major Response Variable

3.5.5.1 Industrial Noise and Blood Pressure

Investigations of noise exposure among occupational groups and in the general population have shown no associations between noise exposure and cardiovascular disease. In general these studies were more methodologically sound, displayed more powerful designs than much of the research previously described, applied inferential statistics to a greater degree and considered several potentially confounding variables in the analysis. The designs represented were historical prospective/paired cohort, historical prospective/cross-sectional and cross-sectional utilizing noise levels and hearing impairment. There were two studies in which noise-induced hearing loss served as a surrogate measure for noise exposure.

A major factor detracting from the studies showing no effects of noise on blood pressure is that of small sample size. Failure to reject the null hypothesis may mean the researcher has not been able to demonstrate that a difference exists or that a difference is probably not large enough to be of practical importance or that the sample size was not sufficiently large to detect differences. Employing too small a sample may result in Type II error which consists of failing to declare that two population mean blood pressures are significantly different, when in fact they are different. The practical control over the Type II error depends upon the investigator specifying what difference is of sufficient importance to detect, the probability he desires of actually detecting it and an appropriate sample size. In general, the probability of committing a Type II error decreases as sample size increases. The issue of sample size and its importance is discussed further in Section 4, p.38 of this report.

Two fairly well designed cross-sectional investigations recently conducted by A. Cohen and colleagues (1980) and Lees and Roberts (1979) showed no evidence of a relationship between noise exposure and blood pressure. Cohen and colleagues (1980a) compared 51 paper mill workers who met high-frequency hearing loss criteria to 51 workers with no more than 20 dB hearing level in either ear for any test frequency. The surrogate noise measure, high frequency hearing loss, used hearing level criteria of 65 dB or more for 3000, 4000 and 6000 Hz. The exposed group averaged 22 years on the job whereas the control group averaged only 12.5 years experience. Blood pressure measures were based on the last two of three readings and were taken by observers without knowledge of the hearing status. The World Health Organization criteria of greater than 160/95 mm Hg was used to define hypertension. Conclusions were derived from a covariate analysis designed to adjust for differences in age and body size. The study suffers from small sample size and possible Type II error. In cross-sectional data of this nature it is difficult to assess selection bias. It is possible that a person who is both hearing impaired and hypertensive is more likely to retire or change jobs than an individual experiencing either problem alone.

The findings of Lees and Roberts (1979) using a similar high frequency hearing loss group and a random sample of controls working in quiet areas of a plant were consistent with those reported by Cohen. There was no evidence of a relation between increased systolic or diastolic blood pressure and hearing loss. Using company health records, persons with hearing loss who worked in high noise areas of a plant were selected for study and stratified into age groups. From these age strata, a random sample of 62 subjects was drawn and matched for age and duration of employment with a control group of 62 persons drawn at random from workers employed in quiet areas. Noise-induced hearing loss was defined as attenuation of hearing greatest at 4000 Hz and at least 20 dB greater than the attenuation at any frequency less than 3000 Hz.

Lees and Roberts do not make clear why such a small sample of subjects was drawn from among the plant employees nor how matching was maintained in the analysis. The failure to detect differences in blood pressure between the hearing loss and control group and the relatively small sample size suggest the possibility of Type II error.

No association between noise exposure and blood pressure was also reported by Malchaire and Mullier (1979) in a cross-sectional study of apparently adequate sample size. Subjects were 1030 car assembly line workers, 581 wire mill workers and 510 individuals never exposed to occupational noise. Noise levels in the car assembly plant ranged from 92-100 dB(A) and 93-97 dB(A) in the wire mills. Although data are lacking as to total vocational noise exposure histories, the exposed workers had been employed at least 3-4 years and hearing thresholds had been determined over a 3-4 year period. The hearing deficit groups had average hearing levels for 1, 2, and 3 kHz for both ears greater than 25 dB. It appears that control group subjects were not assessed for hearing loss. Thus in the analysis, subjects in the two high noise groups were further divided into hearing loss and no hearing loss groups and compared to controls assumed to have no hearing impairment. Although the World Health Organization criteria for hypertension were applied, the authors failed to indicate whether blood pressure was measured pre or post shift and the number of blood pressure readings taken per subject. Twenty-seven Chi square tests applied to the data failed to identify any relationship between exposure to noise as depicted by hearing deficit and blood pressure level. However, according to this project review team, if the data for all the noise exposed subjects are combined and hypertension is defined as blood pressure greater than or equal to 140/90 mm Hg, the results are suggestive of a dose-response relationship; the percentage of hypertensives increases from "the non-noise exposed group" to "the noise exposed but no hearing loss group", to "the noise exposed plus hearing loss group".

3.5.6 No Adverse Effects: Parameters Other than Blood Pressure as the Major Response Variable

Two historical prospective studies and one community survey were identified which showed no relationships between noise exposure and cardiovascular parameters including ischemic heart disease, hypertension, pulse rate, use of anti-hypertensive drugs and consultation rate.

3.5.6.1 Industrial Noise and Health Parameters Other than Blood Pressure

Lees, Smith and Wetherall (1980b) identified 88 employees who had worked exclusively in a low noise area in a production and material handling industry and matched 70 of them with workers exposed to high noise for a fifteen year historical-prospective analysis. Subjects were matched by age (within five years), exposure period and duration of employment. Comprehensive medical records were available for counts of new events of ischemic heart disease, hypertension, myocardial infarction and other illnesses. January 1, 1962 served as a baseline for morbidity measurement. A high noise group was defined as occupationally exposed by job area to prolonged high level noise greater than 90 dBA for a minimum of three consecutive years. A low noise group was represented by those exposed to less than 85 dBA ambient noise for their total work history. Workers exposed at intermediate noise levels were excluded. Thirty pairs of subjects were exposed for 3-6 years, 22 pairs were exposed 7-10 years and 18 pairs for 11-15 years. Work shift which was strongly correlated with noise exposure was considered in the univariate analysis. The study demonstrated no significant differences between the two groups in the incidence of medical conditions for specific exposure periods nor for total period of the study. Unfortunately, as the authors indicate, the sample size was prohibitively small - the increased risk in the noise exposed group would have to be somewhere between three and ten times that of the nonexposed to enable rejection of the null hypothesis. The historical prospective matched-pair design was the major strength of this study yet the matching was not maintained in the analysis. Use of work shift as a potential confounder in a multivariate analysis may have also enhanced the power of this study.

A second historical prospective study, apparently utilizing cross-sectional data from annual examinations of 29 pilots and 29 non-flying control subjects showed no changes in blood pressure, heart rate, cholesterol or glucose levels due to noise exposure (Brown et al, 1975). Pilots and controls had at least eight years in the executive physical examination program and were said to be of similar socio-economic status. Pilots had flown 6000 or more hours. The study suffers from several problems in addition to small sample size. Actual noise exposure levels were not available for the pilots and no noise data were provided for the controls. Comparability of the two groups was not demonstrated and the possibility of selection bias, that is proportionately more healthy than unhealthy men remaining in the program, was not addressed.

3.5.6.2 Transportation, Neighborhood and Community Noise and Health Parameters Other than Blood Pressure

Knipschild and Salle (1979c) found no associations between traffic noise and hypertension, consultation rates, angina or ischemia of the heart in their population survey in the eastern part of the Netherlands. They suggest that the failure to observe a relationship between noise exposure and cardiovascular disease in this ecologic survey of housewives could be the result of a combination of nonresponse, confounding factors such as varying social class among the groups and limited range in level of noise exposure between the two groups. The "noisy" streets had a noise level of L_{eq}=65-70 dB(A) compared to L_{eq}=55-60 dB(A) for the "quiet" streets.

3.5.7 Effects of Short-Term Noise Exposure

Considerable evidence has accumulated over the past three decades from human and animal experiments suggesting that noise may influence blood pressure regulation and other cardiovascular responses. The majority of these studies were conducted in controlled laboratory environments, used noise as an acute stimulus and reported short-term or immediate physiologic responses. Recent and extensive reviews of the experimental evidence of noise effects of the cardiovascular system have been reported by Hattis and Richardson (1980), Kryter (1970), and Peterson(1980).

The goal of the literature review presented herein was to focus on epidemiological studies of long-term exposure to noise and cardiovascular response. However, several experimental studies were included in this review because of their potential for indicating health risk that may exist in real and long-term exposure to noise in daily life. In this report, short-term exposure is used to indicate noise exposure other than life exposure in work and in the general environment which is experienced by individuals over months and years. Specifically, we selected experiments simulating natural environments and/or studies measuring pre-post work shift responses to industrial noise.

Nine studies were identified in the English literature which explored cardiovascular response to short-term noise exposures. Four of these were experiments using healthy young males as subjects and simulated traffic or factory noise as the stimulus. Five were pre-post shift measurements of workers employed in noisy occupations.

Three quasi-experiments using simulated aircraft and traffic noise suggest rather confusing results for short-term noise exposure. Di Cantogno et al (1976) concluded from a non-randomized experiment of 33 subjects and 11 controls of dissimilar ages, that noise may be responsible for an increase in myocardial energy requirements. In randomized experiments exposing 12 healthy males, aged 19-26 years, to simulated aircraft and traffic noise, Mosskov and Ettema (1977a and 1977c) observed an increase in diastolic blood pressure and a decrease of systolic blood pressure.

Mosskov and Ettema (1977b) noted a similar pattern of blood pressure changes when the 12 ostensibly healthy males were presented with simulated textile factory noise while performing mental tasks in a soundproof room. An increase in diastolic blood pressure and respiratory rate and a decrease in pulse pressure and heart rate were observed with the experimental noise load. Subjects served as their own controls and performed sessions in random sequence. It is questionable whether or not the random assignment on noise type to so few subjects adequately controlled for potentially confounding factors. There were differences reported in systolic and diastolic blood pressure, pulse pressure and sinusarrhythmias between the "rest periods of the experiments." This fact alone may mitigate some of the differences observed with noise exposure. The observed decrease in sinusarrhythmias may indicate a decrease in parasympathetic tone, but a decreased heart rate would suggest the opposite effect.

Antonova (1971), investigating the impact of industrial noise on 33 workers in an ore dressing plant, reported noise levels of 92-112 dB in mills and 97-104 dB in separators with relatively high vibration velocities. He observed increases in arterial pressure among mill operators but no change among separator operators when analyzing measurements taken prior to work, 6 hours after beginning the work shift and after work. Too little data are provided the reader as to the quantity and quality of the exposure and health assessments to warrant conclusions from this study.

In a somewhat more rigorous investigation, Dega and Klajman (1977) studied blood pressure and heart rate among men working at propeller grinding compared to similar shipyard workers not exposed to noise. Unfortunately, for comparative purposes, the health outcome measure was reported as heart minute volume according to Starr's formula, a derived parameter based on both heart rate and blood pressure. Since the relationship of this parameter to disease is virtually unknown, the relevance of the outcome measure in the study of noise effects on the cardiovascular system is questionable. The study is of interest because noise levels were plotted in the work sites of the exposed though not the unexposed group; pre- and post-work measurements were obtained on the noise exposed and comparison group of workers; and there is limited, but suggestive, evidence of a dose-response relationship in that ten exposed workers wearing anti-noise ear protectors experienced a decrease in heart minute volume similar to the level in controls.

Cardiovascular vulnerability to intense noise was suggested by Yazburskis (1971) in a study of 36 ostensibly healthy young men working with 8 kc to 20 kc ultrasound for two to five years. Conducting experiments in an ultrasonic laboratory under work conditions, he observed that the noise exposed workers displayed reduced heart rate, enlarged T wave, diminished P and R waves, and reduced systole:diastole ratio. Exercise resulted in a downward shift of the RS-T segment of the ischemic type immediately after work in noise. Blood pressure fell towards the end of the workday; blood pressure did not return to its initial level within five minutes after an exercise tolerance test. These physiological responses are interesting, but of questionable clinical significance. In the opinion of the cardiologist reviewer of this literature, the "junctional" (conjunction) ST depression observed toward the end of the test by Yazburskis is not a clinically significant response.

Measures of 17-ketosteroids and urinary volume have been studied to determine if changes are produced in the presence of noise, assumed to be a stressful stimulus (Gibbons et al, 1975). The acoustic environments of four oil tankers, which were ranked according to noise and vibration values, ranged from 49 to 94 dB. Volunteer officers from the four ships participated in the quasi-experiment. Each officer served as his own control with 17-ketosteroids and urinary volumes determined while on-board ship and exposed to noise and while on leave. The authors concluded that there was a significant decrease in the level of 17-ketosteroid and urinary volume while the officers were serving at sea compared to the values obtained when the men were on leave. Noise could not be assessed independently of vibration in this study aboard oil tankers. Gibbons, Lewis, and Lord (1975) also reported that on the basis of experiments, a decrease in 17-ketosteroids is to be expected with noise exposure and with a combination of noise and vibration. The relationship of this physiological change to overt disease is as yet unknown.

Among the poorer studies methodologically, is a cross-sectional investigation of the free-fatty-acid (FFA) levels in the blood of spinners and weavers. One hundred twenty-one female workers were divided into seven small groups, tested for FFA at varying times during the work day, and compared to nine controls tested before work and in the seventh hour of work. The authors (Proniewska et al, 1972) concluded that cholesterol and beta-lipoproteins increase with a tendency toward FFA rise in the first two hours of work, but at 6-8 hours work there is a considerable increase in FFA with a decrease in other lipid values. Such conclusions are clearly suspect since the data for the exposed and controls are not comparable; cross-sectional differences rather than serial changes over time in the noise exposed workers were compared to serial changes in the small group of controls.

The studies indicating cardiovascular system responses to short-term noise exposure tended to suffer from design and measurement problems. Furthermore, the lack of biologically plausible hypotheses to explain the physiological responses observed reduces the contribution these quasi-experiments might make toward elucidating the health effects of noise exposure.

3.6 Brief Summary of the English Literature

Twenty-six of the 36 studies published in the English literature were cross-sectional in design which provides a weak epidemiological basis from which to infer causal associations. Few of the studies attempted to quantify the observed relationships and only one, Friedlander et al (undated), reported risk ratios. Overall, there was little evidence that other factors which may have contributed to the causal network were studied concomitantly with noise.

The evidence implicating noise as a possible risk indicator is strongest for changes in blood pressure. Seven research groups demonstrated findings supportive of a positive association between noise exposure and blood pressure. Several of these studies provided suggestive evidence of dose-reponse relationships: S. Cohen et al (1980b) demonstrated that blood pressure in children increased with years exposed; Knipschild (1977a) observed higher blood pressures as the level of airplane noise increased in the villages; Parvizpoor (1976) demonstrated an increase in the prevalence of hypertension with increase in length of employment as a weaver. Investigations have also shown no adverse relationships between noise exposure and blood pressure. Two such studies by A. Cohen (1980a) and Lees and Roberts (1979), though more methodologically sound in design than studies showing adverse effects, suffered from small sample size.

Investigations between high noise levels and cardiovascular parameters other than blood pressure provide fragmentary evidence of adverse effects of noise. The most convincing data from the 8 studies reporting some type of adverse effect were those suggestive of dose-response relationships: Knipschild (1977b) found that the contact rate for cardiovascular problems increased with increasing noise level; the Raytheon study reported that workers judged to have always used ear protectors showed the greatest reduction in medical problems while those who never used the protectors showed the smallest changes.

The evidence from the English literature suggests that continued investigation into the effects of noise on cardiovascular responses, especially blood pressure is warranted and that more powerful epidemiological study designs need to be employed in future research.

3.7 Critical Review of the Translated Literature

3.7.1 Adverse Effects: Blood Pressure as the Major Response Variable

Adverse effects of noise on the cardiovascular system were reported in twenty-one of the translated studies. See Table 3-9 for a listing of studies according to the author(s)' conclusions and the ratings of the reviewers. For a study to be replicable and fully informative, both the noise exposure and health variables must be adequately specified. Table 3-9 indicates that most of the studies failed to meet this scientific criterion. Twenty of these studies were cross-sectional in design and, with the exception of three, presented conclusions derived from a large sample of industrial workers.

3.7.1.1 Industrial Noise and Blood Pressure

The investigation of Sanova (1975) was judged by the review team to be one of the more scientifically adequate of these twenty-one reports. Sanova studied 144 compressor operators exposed to continuous noise and 30 controls working in the same plant under low noise conditions. Noise in the compressor shops included 90-110 dB infrasound as well as 87-98 dBA noise levels. All subjects were male, 20-50 years of age, with employment histories from less than one year to more than 20 years. The author gave no information on sample size determination, sample selection, or response rate. Cardiovascular response measures included arterial blood pressure, electrocardiographic readings, contractile blood volume, one minute blood volume, and peripheral resistance. Unfortunately, Sanova failed to provide the reader with definitions and criteria used in determining adverse responses for these health parameters.

Although very little data were presented, the author reported that hypertension rates among the noise exposed group increased and stroke volume decreased with years on the job. In this analysis, it appears that no direct comparisons were made between the high and low noise groups. However, systolic blood pressure was shown to increase more with age among workers

Table 3-9
Studies Ranked by Overall Score, Categorized by Exposure Setting and Nature of Findings* as Reported by Author(s)
Translated Literature

	Scores			
	Noise Exposure	Health Effects	Methodology	Overall Score
I. Industrial Noise - Adverse Effects Reported by Author				
Sanova (1975)	4	3	3	3
Stasiow et al (1974)	4	7	3	3
Britanov (1979)	5	3	2	2
Demeter et al (1979)	2	2	2	2
Geller et al (1963)	2	4	2	2
Graff et al (1968)	2	4	4	2
Jansen (1959a)	2	2	3	2
Pilawska et al (1977)	7	2	4	2
Rumiantsev et al (1971)	2	7	2	2
Cieslewicz (1971)	1	7	3	1
Gel'tishcheva (1980)	2	4	1	1
Kangelari et al (1966)	2	1	2	1
Khomulo et al (1967)	1	7	2	1
Lanzetta et al (1979)	1	8	4	1
Shatalov (1965b)	1	3	2	1
Terentiev et al (1969)	2	1	2	1
Barhad et al (1969)	1	0	3	0
Paranko et al (1974)	1	4	0	0
Tavtin (1976)	3	0	3	0
Vopilkina (1959)	0	4	0	0
Zvereva et al (1975a)	3	1	0	0
II. Laboratory Studies - Adverse Effects Reported by Author				
Quaas et al (1970)	6	9	5	5
Burger et al (1975)	4	9	4	4
Klotzbuecher (1976)	3	4	5	3
Marianiako et al (1975)	3	6	3	3
III. Industrial Noise - Some Adverse Effects Reported by Author				
Ising et al (1979)	6	4	6	4
Folprechtova-Steňzlova et al (1966)	5	7	3	3
Suvorov et al (1979)	3	5	5	3
Kachnyi (1977)	2	6	2	2
Kalicinski et al (1975)	2	4	3	2
Shatalov et al (1962a)	2	3	3	2
Andrukovich (1965)	2	6	1	1
Jansen (1961b)	3	1	2	1
Jirkova et al (1965)	2	1	3	1
Kobets et al (1972)	2	1	3	1
Pokrovskii (1966)	1	2	3	1
Troianskii et al (1971)	1	6	4	1
Zvereva et al (1975b)	3	1	1	1
Shatalov et al (1969c)	1	2	2	1
Capellini et al (1974)	0	6	1	0
Grusha (1974)	0	1	0	0
Kanevskaia et al (1977)	2	0	3	0
Liubashevskaia et al (1976)	0	2	4	0
Shatalov et al (1970d)	0	1	4	0
IV. Transportation Noise, Neighborhood Noise and Community Noise - Some Adverse Effects Reported by Author				
Von Eiff et al (1980)	1	6	6	1
Koszarny et al (1976)	0	1	2	0
Meinhart et al (1970)	0	2	4	0

*A grouping for convenience of reporting.

Adverse effects: the study shows deviations from normal which the author infers to be detrimental to health.

Some adverse effects: the study presents several findings, at least one of which the author infers to be detrimental to health. This category includes studies showing differential effects among population subgroups and/or noise effects for one health outcome but not another.

exposed to noise than among the 30 low noise controls. The clinical significance of the changes in contractile function of the myocardium are essentially unknown. This study, while showing some possible adverse effects, offers little in elucidating an association between noise exposure and high blood pressure. The control group was very small. Furthermore, there was inadequate statistical control of confounding variables and incomplete analysis of the data.

A large-scale cross-sectional investigation of industrial shipyard workers was reported by Pilawska and colleagues (1977) as demonstrating strong associations between noise and health effects. They compared medical record data of 1826 workers exposed to noise levels higher than 85 dB for longer than five hours a day to medical data of 5825 workers from an area where the noise did not exceed 75 dB. Year long and standardized noise measurements were made by dividing the plant into three sections; each section was measured separately using nine daytime and twelve night-time measurement points. The health assessments were less precise, consisting of extant data from periodic examinations conducted by thirteen plant physicians. Diagnostic criteria and definitions of deviations from normal were not specified. Although hearing damage and stomach ulceration were the major disorders identified, the rate of hypertension among the noise exposed workers was twice that of the nonexposed, not taking into account age and length of employment. Sex, history of hypertensive disease and treatment, weight and comorbidities were not taken into account in the analyses. The main strength of this research was the specification of the noise exposure parameter. Unfortunately, its scientific value is limited by inadequate measurement of the health component and incomplete analysis of the data.

Britanov (1979) also reported elevated blood pressure in response to noise exposure in a study of female employees of an acetate and polyvinyl chemical fiber plant. Since the goal of his research was to study the combined effects of noise and acetone, the women were categorized as working in maximum permissible levels of noise and maximum permissible concentrations of acetone; working in noise lower than the permissible level with acetone at the maximum permissible level; and noise levels higher than the permissible levels and acetone at levels three to six times lower than the standard. Unfortunately, Britanov did not include a low noise, low acetone group as a control. Hypertension was defined as arterial blood pressure of 160/95 mm Hg and higher; borderline hypertension as blood pressure between 140/90 and 150/95 mm Hg. The study revealed dominating hypertension and borderline hypertension in the high noise groups (30.0% and 27.5%) compared to the low noise plus maximum permissible level of acetone group (9.7%). The author stated that these data were age standardized, but standardization is not evident to the reader. These observations, although based on prevalence data and not subjected to statistical testing, are suggestive of an association between hypertension and noise exposure. Methodological weaknesses of the study include poor quality control of the measurements, potential self-selection and possible confounding by environmental variables.

In addition to blood pressure, Britanov evaluated the 113 workers using physiological studies, hearing tests and a complaint index. The percentage of persons complaining of cardiovascular disorders was high in the high noise work environments. Alterations of the physiological functions were found to be most pronounced in workers exposed to the simultaneous effect of high noise and acetone.

Cieslewicz (1971) concluded from his study in the spinning and weaving department of mills in Poland that extra-auditory effects of noise represent a serious health problem. His cross-sectional analysis was based on data collected in 1968-69 from 702 weavers exposed to noise ranging from 96-116 dB and 605 spinners exposed at the 84-90 dB level. Hypertension was defined as blood pressure higher than 150/95 mm Hg. He showed a hypertension rate of 35.9% for female weavers over 50 years of age compared to 16.1% for spinners of the same age, sex category; hypertension rates for men (over 50 years of age) were 25.9% for weavers compared to 9.6% for spinners. Although the study is based on a large sample size, it suffers from serious selection bias and failure to control for potential confounders such as age, workshift and social class. In addition, the author failed to provide detailed noise data for the spinners. It appears that both groups of workers were exposed to relatively high noise levels. Although the author attributed the differences he observed to noise, his inferences cannot be defended given the cross-sectional nature of the design and the number of uncontrolled variables.

Another study in the textile industry was so poorly designed and/or described that it is inadequate for judging associations between noise exposure and blood pressure changes (Vopilkina, 1959). However, it is the only study in which the author concomitantly assessed environmental temperature, weight loss of the workers and blood pressure changes during the day. Vopilkina, measuring blood pressures before and after work, found that arterial pressures of spinners and weavers decreased after work, whereas there was little change among the controls. He suggested that these changes could be produced by several factors other than noise such as high temperature. The average weight loss of spinners and weavers in the course of the day, taking water consumption into account, was 1.5 kg. Unfortunately, the data are insufficient for determining the long-term effects of daily fluid loss on the blood pressure of the workers.

3.7.2 Adverse Effects: Parameters Other than Blood Pressure as Major Response Variables

3.7.2.1 Industrial Noise and Health Parameters Other then Blood Pressure

Five studies provide limited evidence of an association between noise exposure and cardiovascular parameters in addition to hypertension. Workers in the oil industry were studied by Geller and associates (1963). In this cross-sectional analysis, 1482 workers in oil gases and 366 in oil gases plus high noise were compared to 263 subjects in administrative work and 456 persons in physical work at the factory. No information was provided as to sample selection or participation rate. Hypotension was defined as less than or equal to 99 maximal arterial pressure or less than or equal to 69 minimal arterial pressure. Hypertension and cardiovascular neurosis were not defined. The workers were said to have been exposed to noise for frequencies from 2400 to 6000 Hz at intensity levels of 115-125 dB. Sex and age were controlled by stratification. Subjects were categorized as under age 40 and over age 40.

Geller and coworkers concluded that arterial hypotension is less common and hypertension much more common in persons exposed to persistent high noise than in workers whose work conditions were not connected with the influence of noise. Cardiovascular neurosis was more frequently found in workers exposed to the effect of noise than in employees performing other physical work in the plant. The major weakness of this study lies in the inadequate information provided on both noise exposure and health outcome including hypertension and hypotension.

In an observational study designed to verify results from experimental noise research, Jansen (1959a) selected workers representative of high and low noise exposure groups in fifteen metallurgical plants in Germany. Among the 1005 subjects, 669 worked in noise somewhat greater than 90 dB and 336 were exposed to noise of less than 90 dB. Jansen defined 34 different jobs within the smelting industry and described the high and low noise groups by job types. The average age of workers in both groups was 42 years. The average length of employment was 11 years; all subjects had worked at least 3 years under the same conditions. Employees lost no pay by participating, resulting in a low refusal rate except for responding to social-psychological questions.

Interviews, physical examinations and blood tests were conducted, presumably by the fifteen plant physicians. Health outcomes included symptoms of vascular and cardiac problems, tachycardia, extrasystole and blood pressure. No diagnostic criteria were given; it would appear that some of the findings labeled "objective" did, in fact, require subjective judgments of the examiner. No blood pressure data were presented.

This was one of the first investigations to describe the high and low noise exposure groups in great detail as to personal characteristics, economic status, family life, living conditions, environmental and working conditions. Although a great deal of data were available and sample size was large, there is no evidence that any of the variables were statistically controlled in the analysis. Nevertheless, Jansen concluded that his study proved that vascular disturbances, skin findings (paleness) and cardiac findings (rhythm disturbances, tachycardia, extrasystoles) are noise determined. It is difficult to concur with such strong inferences for the following reasons: data were not presented in detail with evidence of statistical control of confounding variables; noise exposure and cardiovascular measures were poorly documented; exposure suspicion bias was likely since health measurements were taken by plant physicians, after noise determinations and after talk about work; and each examiner was permitted flexibility in the use of a standardized questionnaire.

Graff and colleagues (1968) studied 117 workers in a boiler plant exposed to 95-110 dBA noise and 50 workers in heavy transportation in the same plant without noise strain. Noise exposure levels for the control group of workers were not provided. It is unclear how these transportation workers differed in noise exposure from the transportation workers described among the noise exposed. Data were collected on blood pressure and other cardiovascular parameters including standard electrocardiography and electroencephalography readings, skin galvanic reflex, and physical examination. Blood pressure was categorized into four grades according to the World Health Organization criteria. No criteria for defining cardiovascular pathologies other than hypertension were given.

The authors observed a larger proportion of hypertensive patients and patients with other heart and circulatory sickness among the noise exposed group than among the workers without noise strain. Unfortunately, detailed analyses by age and length of employment included only the noise exposed group. Within the exposed group, 25-35 year olds, locksmiths and blacksmiths, and individuals exposed at least eight years were most likely to present with hypertension and other circulatory problems. In addition, it was observed that in patients with heart and circulatory disorders and in the group without pathological findings, hard-of-hearing persons were found, but the number of normal hearing persons was lowest in the group of people with high blood pressure. This study provided weak, at best, support for an association between noise exposure and cardiovascular disease.

Three major problems are evident in their cross-sectional data: selective survival and attrition among older workers in the noise exposed group; inappropriate analysis of the data with failure to make comparisons between the noise exposed and the non-noise group; and failure to control for confounding factors. The data were presented in graph form only.

In a recent study of 100 male coal miners, mean age 45 years, Demeter and colleagues (1979) investigated the relationship between hearing loss ("sonic trauma") and arteriosclerosis. They selected 100 male workers and divided them into two groups according to audiometric results. The first group was composed of workers with normal hearing whereas the second group included individuals with "incipient sonic trauma" and advanced hearing loss. Seventy-four of the 100 workers had some type of hearing loss. The authors gave no indication as to how subjects were selected for the potential subject pool from which the 100 were selected. Considering the number of variables studied, the sample size was quite small.

In order to develop an atherosclerosis risk profile, 40 workers (including all hearing levels) were measured for cholesterol, triglycerides, retinal changes, electrocardiographic changes, ponderal index, positive history of smoking, and positive family history. The authors used Feinstein's set theory with the aid of Venn diagrams to analyze the data. They concluded that noise plays an inducing role in arteriosclerosis, that arteriosclerosis favors the occurrence of hearing loss (sonic trauma).

This paper is of interest because of its focus on the interrelationship of hearing loss, arteriosclerosis and noise exposure. However, noise is poorly documented and the data do not appear to be examined in an appropriate fashion. In addition, it is not clear as to what the authors intended to use as a control group. Thus, the correlational data presented do not appear to warrant the strong inferences reflected in the authors' conclusions.

An earlier investigation of the influence of noise on lipid metabolism had also implicated noise in the development of arteriosclerosis. In 1967 Khomulo and associates studied blood serum cholesterol and beta-lipoproteins using standard laboratory methods. They selected a noise exposed group of 69 men and 34 women who ostensibly underwent observation over the course of seven years and a control group of "practically healthy" workers (38 men and 13 women). This design description implied that prospective data were available, yet the analyses showed cross-sectional data only. The noise exposed group had worked for varying lengths of time under conditions of high-frequency noise of 95-117 dB intensity. The control group had been exposed to "permissible" noise of middle and low frequencies with intensity levels from 60-95 dB.

The authors reported that industrial noise of 95-117 dB acting for five or more years, leads to hypercholesterolemia and/or a tendency toward increased quantity of total lipids and beta-lipoproteins in the blood. It was further observed that the degree of increase in cholesterol depended upon the length of employment in conditions of intense noise. This observation is suggestive of a dose-response relationship since a comparable increase in cholesterol did not occur in the control group. The strength of this study lies in the standard assessment of blood lipids. However, the fact that data are not presented to support other than a cross-sectional strategy limits conclusions which might be derived from these data.

3.7.3 Adverse Effects: Steady versus Non-Steady Noise

Several studies in the translated literature compared effects of steady and non-steady noise. In studies described elsewhere in this literature review, the investigators did not indicate that specific comparisons were made between noise exposures which were steady or non-steady. In many cases the noise source and type were not described. For purposes of this review, the term "stable" was interpreted as steady according to the *American National Standard Methods for the Measurement of Sound Pressure Levels* (American National Standards Institute, 1976).

Shatalov (1965b) observed that in persons exposed to continuous noise, symptoms of vascular dysfunction occurred. Vascular dysfunction was defined as lability of the arterial pressure, tendency toward the reduction of venous pressure and the reduction of peripheral resistance, and bradycardia. Under exposure to intermittent noise, there was a tendency to hypertension. Shatalov did not specify the occupational groups studied and failed to use a control group of individuals exposed to low noise levels. Subjects were classified according to continuous noise and intermittent noise exposure; 368 individuals worked in continuous noise of 85-112 dB and 221 subjects worked in intermittent noise of 85-111 dB. Individuals with a history of cardiovascular disease were excluded. In the group of 589 workers, 23% had been employed approximately 5 years, 24% from 5-10 years and 53% for more than 10 years. It is unclear as to how length of employment was used in the analysis. No information is given as to sampling frame, assemblage of the study groups nor response rate.

In addition to the above analyses, blood pressure effects were studied in 1357 persons; 1019 exposed to continuous noise and 338 to intermittent noise. Noise levels and frequency composition of the noise were not described for the 1357 persons. Blood pressure measures were taken at the beginning of the day, after a 10 minute rest. High blood pressure was defined as values greater than 130/90 mm Hg for persons less than 40 years of age and values greater than 140/90 mm Hg for workers over 40 years of age. Low blood pressure was defined as readings less than 100/60 mm Hg. Shatalov also studied several other hemodynamic indicators but failed to report diagnostic criteria or measurement procedures for them: electrocardiography, ballistocardiography, phonocardiography, peripheral resistance, venous pressure, heart size.

Virtually no raw data are provided the reader. Consequently the cardiovascular effects reported by Shatalov cannot be evaluated adequately. In the analysis of blood pressure data, there is no evidence of controlling for two strong confounding variables, age and sex. Although the author may have considered this study supportive of an adverse relationship of noise exposure to cardiovascular disease, the reviewers judged the study to contribute little, if anything, to the understanding of that relationship because of poor design and questionable clinical significance of some of the measures.

The importance of differentiating between "stable" (steady) and intermittent noise was demonstrated by Mariniako (1975) in a quasi-experimental laboratory study of twenty healthy men. Subjects were exposed to four series of noise: low frequency steady noise; low frequency noise with intermittent effects similar to work in concrete packing; high frequency steady noise; and intermittent noise similar to hydraulic testing of pipes. In each series, subjects were exposed to noise for a total of one hour as follows: Series 1, steady noise; Series 2, noise presented for 2.5 minutes alternating with 5 minute intervals; Series 3, steady noise; and Series 4, 0.5 minute noise alternating with 0.5 minute pauses. The findings suggested an adverse effect from noise with intermittent sound leading to a greater degree of vessel vasoconstriction than steady noise.

Another cross-sectional study of workers exposed to stable and pulsed noise was conducted in a machine building plant (Tavtin, 1976). A total of 861 workers, 281 women and 580 men, were divided into five noise exposure groups. Group 1 was composed of 121 workers in 70 dB noise; Group 2 of 139 workers in 83 dB noise; Group 3 of 168 workers in 94 dB noise; Group 4 of 267 workers in 110 dB noise; and Group 5 was composed of 166 workers in 114 dB noise. Cardiovascular dysfunctions were assessed by clinical examination. No diagnostic criteria or definitions were provided the reader. No actual data were presented. However, the author reported that under exposure to continuous noise, there was a significant increase of cardiovascular system disorders (6.3%). Under exposure to pulsed noise of 114 dB, functional disorders of the cardiovascular system occurred in 8.4% of the subjects. Tavtin concluded that a significant increase of functional disorders of the nervous and cardiovascular system takes place under exposure to noise of a general level of 110 dB and under exposure to pulsed noise of 114 dB. Unfortunately, there is no indication that observations in the high noise group were compared to the low noise group. In addition, this cross-sectional study provides no analysis in which age and sex, strong potential confounders, were controlled. Thus, the reviewers concluded that the data presented are inadequate to support the author's conclusion of a significant increase of cardiovascular disease under exposure to noise.

3.7.4 Some Adverse Effects: Blood Pressure as the Major Response Variable

3.7.4.1 Industrial Noise and Blood Pressure

Several of the 19 studies grouped as showing some adverse effects are fairly rigorous in design and suggest that noise exposure may be positively associated with cardiovascular responses. Observations by Folprechtova-Stenzlova and Janicek (1966) were suggestive of an association between noise and blood pressure in that foundry workers with longer exposure to noise (in years of employment) had higher blood pressures than workers with short exposures. As the authors point out, age was only partially controlled in the analysis, so these data must be interpreted cautiously. Different levels of noise did not affect the level of blood pressure when years employed and schedule were held constant. However, all noise levels were high; the average noise level for the high exposure group was 108 dB compared to an average of 92 dB for the low noise group.

One of the unique features of the Folprechtova-Stenzlova study is its consideration of potential cardiovascular risk factors including weight, fluid intake, salt intake, fat intake, smoking history, sleep patterns as well as the stress of commuting, work schedules, and number of children. Lower values of average blood pressure were found in workers commuting to work, those with relatively low body weight and those limiting fluid intake. Since these variables were evaluated only as single indicators, it would be informative to subject the data to multivariate analytical techniques currently available to explore the contribution of noise while adjusting for other environmental influences on blood pressure.

Kalicinski and colleagues (1975) reported a dose-response relationship between noise and hypertension similar to that observed by Folprechtova-Stenzlova and Janicek (1966). The frequency of hypertension increased from 26% for women employed for 1-6 years to 47% for women working 13 or more years in noise. These investigators studied 140 women working in the spinning and weaving industry who had been exposed to similar noise levels for varying periods of time. The noise ranged from 95-105 dB for frequencies of 32 to 16,000 Hz. The subjects ranged in age from 47-51 with an average age of 49 years. Hypertension was defined as blood pressure greater than 140 mm Hg systolic and greater than 90 mm Hg diastolic.

In addition to blood pressure, symptoms of coronary disease and inadequate blood supply to the heart were evaluated. The frequency of symptoms of inadequate blood supply to the heart muscles indicated by changes in the S-T segment on electrocardiograms was greater, the longer the occupational exposure to noise. Since the S-T changes occurred most often among those with high blood pressure, they may represent changes due to hypertension rather than independent signs of coronary disease. These dose-response relationships are also questionable because of potential selection bias, inadequate measurement of noise exposure and the failure to control for confounders other than sex and age.

Five other studies showed blood pressure effects of noise. In a cross-sectional study of 300 female weavers working in noise of 102-108 dB, Kachnyi (1977) observed a preponderance of hypotensives at the shorter years of service. However, the number of hypertensives, although very low in this young population, increased with the length of employment, suggesting a dose-response relationship between noise exposure and hypertension. Hypotension was defined as blood pressure less than 100/55 mm Hg and hypertension as greater than or equal to 140/90 mm Hg. For no apparent reason, the researcher took the arterial pressure in the middle of the first shift of the last day of a five day work week.

The strengths of this study were in the size of the population and the selection of subjects with no industrial noise exposure other than that under investigation. The research is not very useful for judging the association between noise and blood pressure because comparisons were not made with the control subjects.. There was limited use of analytical techniques with no evidence of statistically controlling for age, obesity, family history of hypertension or other stressors which may have biased the results.

An earlier study from a population with remarkably low mean blood pressure levels provides data of limited usefulness. Andrukovich (1965) compared the blood pressures of 846 spinners and weavers in a textile factory to the blood pressures of approximately 8972 women in the general population. He found that arterial pressure was higher in female weavers compared to the mean age-specific indices for arterial pressures in women of the control population. The differences observed were statistically significant for the age groups 16-19, 30-39, and 40-49 years.

While this study enjoyed a large sample size, there appears to be a strong potential for selection bias. The noise levels in the spinning sections of the factory ranged from 87-88 dB and in the weaving sections from 99-102 dB. There was no indication that the general population controls were screened for noise exposure and no information as to how the age-specific blood pressures were obtained. The blood pressure data of the workers were collected over a period from 1959-1963. It is unclear as to how multiple blood pressure readings per person were used in the analyses.

Jirkova and Kremarova (1965) investigated the effect of noise on the general health of workers in large engineering factories. Their data are suggestive of a positive association between hypertension and noise levels. However, the differences observed were not statistically significant. The observations were based on 766 men and 203 women from noisy work places compared to 371 men and 318 women from not-noisy work places. Data were collected from the records prepared routinely by plant physicians with no information as to quality control or criteria applied in abstracting health information. High noise ranged from 85-115 dB among 34 plants studied. The quieter areas had noise levels lower than 70 dB. The authors indicated that in the noisy work places the noise was considered disturbing because its intensity exceeded the amount necessary for conveying information whereas in the quiet areas the noise level was not disturbing. The major weaknesses of this study were the poor noise exposure data provided, the lack of quality control of the health outcome due to the use of medical records, and the failure to control for potential confounding variables. The data were not examined in a multivariate mode.

Shatalov, Ostapkovich, and Ponomareva (1969c) studied hearing and arterial blood pressure in 806 persons exposed to 90-122 dB broad band noise of high frequency and 210 individuals of respective ages with normal hearing not exposed to industrial factors. Workers were selected from the needle section of a ballbearing plant, and twisting section of a fiber production plant. The work conditions of the control group were not stated. Subjects were categorized by hearing group as unchanged hearing, slight hearing loss, moderate hearing loss and severe hearing loss. No subjects were classified with severe hearing loss.

The authors observed that in men working in conditions of noise, regardless of age and degree of hearing loss, the systolic blood pressure was substantially higher than in men of the same age in the control group. The exception was for men younger than 40 years of age with moderate hearing loss. Among women less than 40 years of age working in noise, an increase in systolic and diastolic blood pressure was noticed regardless of the hearing loss category. In subjects of comparable age and sex, no variation in blood pressure in relation to degree of hearing loss was found. The authors concluded that for persons working in conditions of intense industrial noise changes in arterial blood pressure precede hearing damage. Therefore, the role of vascular disorders in the development of occupational hearing impairment cannot be excluded.

This cross-sectional study included inadequate information on the assemblage of the noise exposed and control group, too few control subjects, insufficient information on sound and blood pressure measurement, and incomplete control of confounding variables. It was judged by the reviewers to be too poor to support the conclusion that an association between noise and blood pressure exists. A major problem of a study of this nature is that it is difficult to determine the extent to which selection bias may be operating due to removal of individuals with both hypertension and hearing loss from the work force.

Another study by Shatalov and Murov (1970d) demonstrated that exposure to noise and emotional tension led to an increase in hypertensive disease. In their study, 2034 men and 1896 women were grouped by noise exposure. Group 1 was composed of 1275 fitters, loaders and lathe operators exposed to high frequency noise of 95-112 dB; group 2 consisted of 339 operator-testors with noise analogous to group 1 and in work associated with neuropsychic tension; group 3 consisted of 1172 scientists involved in mental work associated with tension but no noise; group 4 consisted of 1144 technicians and skilled mechanics in work not connected with noise nor tension. No information was provided as to sampling frame, determination of sample size, sample exclusions and/or nonresponse. No information was given regarding the blood pressure measurement procedures or conditions under which the readings were taken. Age and sex were controlled in the analysis with stratification and standardization procedures.

Mean systolic and diastolic blood pressures and rates of hypertension were reported. Multiple t-tests were used to compare the age/sex groups on mean systolic and diastolic blood pressures. There apparently was no statistical control of factors such as family history of hypertension, medications, exercise, smoking history, or diabetes. In addition, no information was provided relative to the hearing thresholds of subjects nor the duration of employment. The authors interpreted their data to suggest that noise adversely influences blood pressure; and that noise and tension in combination influence hypertensive disease to a greater degree than either operating alone. The study design and analytic methods were weak for drawing such inferences.

3.7.4.2 Transportation, Neighborhood and Community Noise and Blood Pressure

The findings of one community survey were suggestive of an association between noise exposure and hypertension. Von Eiff and Neus (1980) explored the feasibility of studying the impact of traffic noise on the cardiovascular health of residents in Bonn, Germany. A high noise area was defined by a noise level of 66-73 dB(A); a low noise area by a constant noise level of a maximum of 50 dB(A). A random sample of 458 men and 473 women between 20-59 years of age was contacted by letter and later interviewed in the home. Only five persons failed to respond. Aliens, residents of less than three years and families with apartments above the second floor in the noisier areas were excluded.

High noise area residents more often than low noise area residents indicated existing hypertension or hypertension under treatment. Age and sex were controlled in the analysis by stratification. The groups differed on social class and smoking. Hypertension treatment was not found to be associated with alcohol, coffee or tea consumption, home ownership, smoking or income per capita. The authors concluded that a prospective epidemiological investigation was justified.

This cross-sectional survey was one of the better studies reviewed although it specified the noise parameter poorly and was ecologic in that individual hypertension status cannot be linked with level of noise exposure. It could have been strengthened by having blood pressure readings conducted at the time of interview.

3.7.5 Some Adverse Effects: Parameters Other than Blood Pressure as Major Response Variables

3.7.5.1 Industrial Noise and Health Parameters Other than Blood Pressure

A recent and methodologically rigorous study was conducted by Ising and colleagues (1979) to test methods that might be useful in studying noise effects relevant to health and to explore the relationship of noise to cardiovascular disease risk. It was the only study identified for review which utilized dosimeters to estimate personal noise dose. It also provided detailed description of the health measures and applied inferential statistics to the data.

The study was designed to include both cross-sectional and interventional strategies. Of 100 employees of a brewery who were invited to participate, 90 volunteered. Six work environments within the plant were evaluated for noise using precision sound level meters and measurements in compliance with ISO standards. In addition, each subject wore a dosimeter on the upper body during the investigation. The mean noise level and standard error for the 36 noise exposed workers was 95 ± 0.7 dB(A) and for the 54 controls was 82 ± 1.2 dB(A). A subgroup of 30 subjects exposed to noise and 16 controls were examined during work. The controls were studied for one day, whereas 18 of the noise exposed workers were examined for two days and 12 of them for two weeks. These 12 subjects wore hearing protectors half of the time and worked without hearing protectors the other half of the time.

Medical parameters studied included blood pressure, total cholesterol, potassium, total protein, glucose in the urine and biochemical parameters such as norepinephrine, epinephrine, vanillyl mandelic acid and creatinine. In addition, data were collected on age, body height and weight and hearing thresholds. Blood pressure was measured at the end of the work shift with a semi-automatic measuring device. The mean value from a minimum of four blood pressure readings was recorded as the individual's blood pressure.

The investigators found that the systolic blood pressure and the excretion of vanillyl mandelic acid and noradrenaline were higher by 7 mm Hg, 67% and 16% respectively, when subjects worked without hearing protectors at a mean noise exposure of 95 dB(A) than when they worked with hearing protection. The actual daily average noise level reduction of the Bilsom capsule protection device was 13 dB. Working without hearing protectors reduced the magnesium concentration in the blood. Magnesium concentration was negatively correlated with increase in blood pressure when exposed to noise. The authors also concluded that only the examination of the same test subject under different noise conditions is suitable for studying noise effects. Their comparison of blood pressures of noise workers and a control group indicated no statistically significant differences. However, the difference observed was suggestive of a noise effect. Given the small sample size, one would not expect to observe strong blood pressure effects in these cross-sectional observations.

Although the Ising et al study (1979) was judged to be methodologically sound, the reviewers cannot agree with the suggestion that an external control group is not needed because this jeopardizes the internal validity of the study. The reviewers would agree that intra-individual as well as inter-individual differences need to be considered in study designs. In addition, the data appear to be inadequately adjusted for age, sex and weight. Statistical control of several other potentially confounding variables such as smoking, comorbidities and family history of hypertension would have enhanced the study. This pilot project indicated the usefulness of intra-individual as well as inter-individual values for investigating some but not all cardiovascular parameters, proposed an intervention model from which some cardiovascular risks could be estimated, and demonstrated the feasibility and value of estimating personal noise dose. Application of the proposed techniques in studies of reasonably large populations to test the influence of long-term and varying noise exposures on the development of overt disease is yet to be demonstrated.

A fairly well designed study of workers in the machine building industry provides some evidence of a positive and dose-response relationship between noise exposure and neurocircular impairment (Suvorov et al, 1979). The evidence of an adverse relationship between blood pressure and noise is weak, although there appears to be such a trend in the data. Suvorov categorized workers by degree of noise exposure: Group 1 included 121 office employees working in noise levels averaging 70 dBA (control condition); Group 2 included 139 turners with noise exposure of 84 dBA; Group 3 included 168 cutters with noise exposure of 93 dBA; Group 4 consisted of 267 motor mechanics exposed to noise at the 100 dBA level; and Group 5 was composed of 166 punchers exposed to noise levels of 115 dBA. The cross-sectional sample included 587 males and 274 females, average age 34-38 years, and average length of employment 11-16 years. Hypertension was defined by standard criteria as greater than 140/90 mm.Hg, hypertensive disease as 159/94 mm Hg and hypotension as less than 100/60 mm Hg. Hypertensive neurocirculatory asthenia and atherosclerotic cardiosclerosis were determined by medical examination. No measurement procedures, quality control of the data or indication that the therapists were unaware of noise exposure status of the workers were given.

A regression analysis indicated an increase in neurovascular impairment by 0.5% with each increase of 1 dBA in the level of noise. The rate of hypertensive disease in workers under noisy conditions was higher than in the office workers exposed to 70 dBA noise, but these differences were not statistically significant. Suvorov and colleagues also concluded that at low noise levels neurovascular disorders prevail, while hearing losses prevail at high levels. This suggested to the authors two different, but related, mechanisms in the action of noise upon the worker. The major methodological concern in this study was the incomplete control of confounding variables, especially age, sex, weight, and history of disease. Unfortunately, these prevalence data do not allow one to infer causal associations since the temporal relationships of noise and neurovascular impairment and/or hypertension were unknown.

Capellini and Maroni (1974) investigated eleven risk factors for cardiovascular pathology in relation to work activity in a chemical industry near Milan. In this cross-sectional analysis, 1286 men and 60 women, representing 98.6% of the factory employees responded. Each homogeneous age group was evaluated for disease frequency as well as assessed on characteristics of the environment including noise, stress, physical exertion, vibration, work schedules, temperature, lighting and global exposure to toxic substances. Hypertension was defined as systolic blood pressure higher than 160 mm Hg and diastolic blood pressure greater than 90 mm Hg. Coronary disease included progressive angina pectoris and myocardial infarction which was diagnosed by specialists on history and/or from electrocardiograms. Although the investigators reported intense noise on the order of 85-95 dBA, no specific information is provided as to noise parameters or subject criteria.

The frequency of hypertension by age groups was not significant for any of the eleven factors studied. The authors concluded that the risk for coronary disease due to exposure to intense noise was equal to that in the non-exposed population by an increase in age of ten years. That is, using a weighted regression analysis it was observed that the probability of coronary disease in noise exposed workers of a given age group was nearly identical to that of non-exposed workers in the next age group. Unfortunately, there is no indication that blood pressure and sex were considered in the analysis. The noise exposure variable is so poorly described it is difficult to evaluate this research. The strength of the study lies in the verification of the diagnoses of cardiovascular disease.

A comparative study of 1005 persons employed in the smelting industry in Germany showed no defined noise disease, but suggested that vegetative disorders including peripheral circulatory symptoms and cardiac signs and symptoms (tachycardia, rhythm abnormalities and extrasystoles) may be related to work in noisy environments (Jansen, 1961b). These conclusions were based on very subjective sign-and-symptom data with no evidence of reliability or validity of the assessments. Therefore, this cross-sectional study was judged to be of little value to the assessment of cardiovascular effects of noise.

One study was identified which explored the relationship between noise and blood serum cholinesterase (Troianskii et al, 1971). Cholinesterase is an enzyme that destroys acetylcholine, a neurotransmitter. The subjects were

55 specialists working in diesel and blower stations, 16 working 12 hours with 24-hour breaks; 24 persons with 12-hour breaks; and a control group of 15 persons working under similar conditions without noise. The noise was said to range from 94-97 dB at medium and high frequencies. The activeness of the cholinesterase was determined according to Hestrin's method, before work, after three hours of work and at the end of each shift. Among the noise exposed subjects there was a significant lowering of cholinesterase with increase of hours worked, suggesting a dose-response relationship. However, the lack of a three-hour measurement in the control group makes complete comparison impossible. Blood pressure was also studied with no association with noise exposure noted. Apparently the relationships between noise, blood pressure and cholinesterase levels were not investigated.

Troianskii et al gave no information on comparability of the groups, environmental exposures other than noise or existing disease in the workers. The authors interpreted the lowering of the activeness of cholinesterase as an indication of parasympathetic dominance in persons working under the influence of noise. However, there is no known evidence that a fall in cholinesterase has any long-term pathological significance relative to cardiovascular disease. This research by Troianskii and colleagues is believed to be among the first to suggest a relationship between cholinesterase and noise and probably deserves further evaluation regardless of its potential influence on the cardiovascular system.

3.7.5.2 Transportation, Neighborhood and Community Noise and Health Parameters Other than Blood Pressure

Several community surveys exploring cardiovascular effects other than hypertension were found to contribute little, if at all, to the evidence for an association between noise exposure and health. From a cross-sectional survey of 256 residents living in a zone with noise greater than 100 dB(A) and 255 residents of an area with a noise level of 80-90 dB(A), Koszarny and others (1976) concluded that the relationship between some symptoms of aggravation and ill health and the acoustic conditions in the place of residence, indicated the probability of the negative influence of airport noise on the resident's state of health. The percentage of persons complaining of symptoms including heart pain, nervousness, and of taking cardiac medicine was higher among women living in the worst acoustic conditions than among men. Unfortunately, the authors failed to report how they obtained their airport noise area data and their symptom data. In addition, it is unclear as to how comparable and equal numbers of subjects were selected from the two areas without introducing selection bias. Overall, this study contributes little to the understanding of the relationship of noise to health effects.

In a general community survey, Meinhart and Renker (1979) compared 807 males with noise impaired hearing to the morbidity statistics of 3,948 from the local health clinic of the same district. Data were obtained from medical records ordered by the labor sanitary inspection and from record data of an ambulatory clinic. Six catagories of cardiovascular response were noted: all circulatory diseases; functional heart and circulatory disease; coronarsclerosis and myocardial injury; hypertension; hypotension; and peripheral vascular disturbances. The authors reported the prevalence of myocardial injuries for the noise-impaired hearing men to be twice that of the normal population. The prevalence ratio of hypertension between the noise impaired and controls for 15-40 year olds was 7.6; for 65 year olds, 9.7; and for 65+ year olds, 8.2. For the younger age groups hypertension was higher among the noise impaired than among the clinic population. No differences were observed between the groups for functional heart and circulatory diseases and peripheral vascular disease. A dose-response relationship was reported on the basis of length of employment. For all age classes the frequency of heart diseases and especially hypertension and hypotension began rising after five years employment and rose precipitously after 20 years of employment. It should be noted that age effects were not taken into account in this analysis. No data were provided as to control of variables such as co-morbidities, medications, and treatments. From the data presented in tabular form, co-morbidities were observed in both groups. Although blood pressure probably changed with onset of myocardial injuries and/or treatment, no attempt was made to control for blood pressure levels in the heart disease data analysis. The study conclusions are also questionable because of the strong possibility of selection bias.

3.7.6 Some Adverse Effects: Steady versus Non-Steady Noise

Several studies comparing steady and intermittent or pulsed noise effects have reported findings indicating that both the character of noise and individual characteristics are important. Pokrovskii (1966) reported that the systolic blood pressure of 17-30 year old workers exposed to high frequency pulsed noise of 90-95 dB was lower than comparable age workers exposed to medium frequency "stable" (steady) noise at 80-85 dB, whereas older workers exposed to the high levels showed systolic blood pressure higher than controls. Changes in blood pressure during noise exposure were higher in persons with symptoms of hypotension or hypertension than in persons with normal blood pressure. These blood pressure changes were more pronounced in persons exposed to pulsed noise than to steady noise. Brachycardia or tachycardia occurred on the average five times higher for persons exposed to pulsed noise than for the controls.

Pokrovskii's cross-sectional study included 408 men in the machine building industry exposed to steady noise of 80-85 dB compared to 587 workers exposed to pulsed noise of 90-95 dB. One hundred twenty of these subjects were studied for changes in blood pressure during noise exposure. The study is too weak methodologically to permit inferences. It suffers from selection bias, uncontrolled confounding factors and poor documentation of the noise exposure.

More recently, Kanevskaia et al (1977) investigated the effect of both steady state noise and pulsed noise on the sympatho-adrenalin system. The study included 256 workers exposed to "stable" noise at levels of 90-100 dBA, 284 workers exposed to pulsed noise at levels of 107-117 dBA, and a control group of 100 workers who supposedly were not exposed to noise exceeding the maximum permissible level. The author collected information on age, length of employment, sex, and work environment but failed to control for any of these in the analyses. Multiple outcome measures were used ranging from blood pressure to dermographism, adrenalin and noradrenalin content in the urine, hearing thresholds, reported complaints such as headache and irritability, and skin vibrational sensitivity. Unfortunately, these authors provide no diagnostic criteria, definitions or measurement procedures for the health outcome.

The adrenalin content in one-hour urine of persons exposed to "stable" noise was higher whereas the noradrenalin was slightly lower than in the control subjects. For workers exposed to the effect of pulsed noise, adrenalin content in the one-hour urine was greater than with "stable" noise, but the noradrenalin was similar to the control values. The authors also reported, but presented no data, that blood pressure was elevated in 35% of the workers exposed to pulsed noise compared to 22% in the "stable" noise group. It is unclear as to what extent the control group of 100 were assessed and used in the comparison on the multiple outcomes since no data were provided in the paper. Unfortunately, this study offers little in the search for associations between noise and cardiovascular effects.

The design and health outcomes were so poorly described in a study of noise in a milling plant, that it commands little comment (Zvereva et al, 1975b). Workers exposed to the effect of intermittent noise were evaluated before the beginning of the work shift for hearing thresholds, arterial pressure, and pulse rate. No data are provided the reader and no quantification of the blood pressure responses are given. The authors concluded that complaints in many cases were combined with objective symptoms of disorders of arterial pressure, more often elevated pressure and pulse lability.

3.7.7 Effects of Short-Term Noise Exposure

The translated literature provides somewhat more evidence of short-term noise effects on the cardiovascular system than the literature on short-term effects published originally in English. In addition to the research of Ising et al (1977) and Troianskii et al (1971) previously described, nine studies reported effects of short-term noise exposures.

Stasiow et al (1974) studied the effect of 7.5 hours of noise exposure on the cardiovascular system. Thirty-one workers in a mechanical coal processing section of a mine, aged 35-62 years, and employed for more than six years participated. No control subjects were used. Blood pressure, electrocardiographic readings, cold pressor tests and retinography measures were taken before work after a night's rest and repeated after 7.5 hours of work in noise ranging from 86-100 dB. The electrocardiograms were evaluated according to the Minnesota code. Stasiow and colleagues concluded that under the influence of exposure to industrial noise, a significant increase in the diastolic pressure and a spastic state of the peripheral arterial vessels occur. They also reported that exposure to industrial noise caused an increased vessel reactivity and changes in the electrocardiographic curve which they believed were related to domination of the parasympathetic system. However, without background information on the health state of each subject, it is difficult to judge the clinical significance of the reported electrocardiographic changes. This one-group before-after design is weak in controlling for confounding extraneous variables. Furthermore, an investigation of the cardiovascular responses to a one day exposure to noise in individuals routinely working in noisy environments, provided insufficient information for inferring to long-term noise effects.

Gel'tishcheva (1980) studied the dynamics of the functional state of the cardiovascular system in adolescents performing delicate visual work involving industrial noise (the assembly of men's wrist watches). The study, quasi-experimental in design, assessed multiple cardiovascular indicators during the course of one day, one week, and at the end of one year. Thirty-six adolescents, aged 16-18 observed under industrial work conditions, were compared to a control of 11 students, aged 16-17, undergoing industrial training and working at their own tempo. Subjects were said to be healthy and of average physical fitness. No differences were noted in the cardiovascular indicators by area, age, length of employment, or task performed. There were also no differences in the initial value of the cardiovascular indicators between the adolescent workers and the control students. Actual exposures to noise were not stated. Apparently, both groups of subjects were exposed to similar noise conditions not higher than 75 dB. The health outcomes measured were blood pressure, electrocardiographic readings, and pulse rate.

The author concluded that "the intensive, delicate visual work performed under exposure to occupational noise not exceeding 75 dB shows an adverse effect on the functional state of the cardiovascular system in adolescents". However, it should be noted that the observed changes over the course of the year of work in noise (drop in systolic pressure, systolic blood volume, minute blood volume, and rise in diastolic pressure; reduction in frequency of heart contraction and increase of the projections R and T of the electrocardiograms) occurred in both the adolescent workers and the control group.

Since both workers and student controls were exposed to similar noise levels, the detailed findings regarding the state of the cardiovascular system cannot be adequately assessed without a more appropriate comparison group, that is, a group of subjects exposed to a different noise level. This study also suffers from small sample size and poor specification of noise and cardiovascular parameters.

In yet another poorly designed study, the authors concluded that a one hour per day exposure to noise in the 110-112 dB range can be successfully endured, but that daily exposures of 3 and 6 hours will bring about harmful effects (Terentiev et al, 1969). For noise levels in the range of 112-120 dB, severe reactions were noticed among 90 workers exposed to aircraft noise. The reactions observed were that the frequency of the heart contractions decreased, systolic and diastolic blood pressure decreased, as a rule; the beat and minute volume of the heart decreased markedly; and the reaction of the cardiovascular system to physical exertion increased. Furthermore, during daily exposures of 1, 3, and 6 hours of noise at 120 dB an increase in systolic and diastolic blood pressures was observed. Although these findings are consistent with other reports in the literature, they must be viewed with caution since no controls were employed, before-after work data were inadequately analyzed, confounding variables were apparently not considered, and data were not presented to support the conclusions. Before-after work measurements and measures taken at monthly intervals for unknown periods of time were supplemented with anecdotal notes to produce Terentiev's results.

Lanzetta and colleagues (1979) reported an increase in the cardiac rate in response to short-term noise exposure. They studied serial electrocardiographic measurements of 12 workers exposed to noise of two different work environments and 5 workers not exposed to substantial noise. The Holter electrocardiographic monitoring method, a continuous ambulatory electrocardiogram made by tape recording for analysis at a later date, was used. Of the exposed workers, 8 had normal hearing and 4 had hearing impairment; the controls had normal hearing. The study began 3.5 hours after the start of the work day and ended 2 hours after the end of work, thus including 4.5 hours of unbroken exposure to noise and 2 successive hours of rest for each subject. The authors reported that the increase in the cardiac rate was constant in the group exposed to noise with normal hearing, was maintained throughout the work shift and reentered the normal limits only after cessation of work. The results were statistically significant in comparison with the group of workers exposed to noise less than three years, the recovery of the base cardiac rate occurred within 44 minutes; for those exposed 4-21 years, the recovery occurred between 58 and 120 minutes; no linear relation between recovery time and age of the worker was noted. Unfortunately, the methodology employed in this study of short-term effects of noise was described very briefly. Multiple potential confounding variables were considered in data collection, but the extent to which they were employed in the analysis is not clear. The Holter electrocardiographic monitoring method may be useful in future studies if combined with concomitant noise level monitoring.

Another study of short-term effects of noise was conducted by Barhad et al (1969) among workers at a large iron works factory. A cross-sectional pre-post shift design was employed to test 132 workers exposed to noise during the working day. No non-exposed controls or replications of the study under non-noise conditions were used. The noise frequency was reported to include a spectra extending over several octaves with intensities between 87-127 dB. Little information is provided relative to the exposure status. Apparently 100 of the 132 workers had been employed more than five years. The authors concluded that most of the workers showed, toward the end of the work day, a decrease of systolic and diastolic pressure which varied between 7 and 14 mm Hg compared to the values found at the beginning of the work day. Smelting division workers showed a significantly higher disease rate than workers in the motor section of the plant. The study design and data analysis are weak for judging either short- or long-term effects of noise.

In an early study of two groups of workers experiencing relatively high noise levels, Shatalov and colleagues (1962a) examined hypertensive and electrocardiographic effects of noise. Studying 300 subjects, 156 workers in a twisting plant exposed to 85-95 dB noise and 144 workers of a ballbearing plant exposed to 114-120 dB of noise, they failed to observe the hypertension effect of noise reported in the literature. Workers often experienced labile arterial pressure, bradycardia and nonspecific T-wave changes, especially after physical stress and at the end of the work day. In this study, there were some 80? men and 120 women; 225 who were younger than 40 years of age and 143 who had worked in noise ten years or more. Unfortunately, the authors failed to control for age, sex, and duration of employment in the analyses. Since an unequal distribution of men and women in the twisting and ballbearing plant is highly likely, these results must be interpreted with caution.

Liubashevskaia and Solonin (1976) attempted to study the effects of improvement in the work environment of 16 forge workers and 12 apprentices. Before improvements, the general noise level was 95 dB average; after improvements it was 90 dB average. They concluded that systolic and diastolic blood pressure decreased at the end of the shift and that blood pressure decreased as temperature in the work area increased. Their conclusions are not supported by the data.

A quasi-experimental study of eight clinically and otologically healthy males, 22-35 years of age, demonstrated that the use of the ear protectors did not prevent the cardiovascular influence of noise at the 90 dB level (Quaas, 1970). In this study, three single tests on each subject were performed with the sequence of the tests permutated. Unfortunately, the subjects knew the respective sequence of the tests which could have influenced the results. Under test one conditions, subjects using ear plugs spent 10 minutes at rest, 30 minutes at continuous exercise followed by 10 minutes of rest; under test two conditions, subjects wearing ear plugs, spent 10 minutes at rest, 30 minutes at continuous exercise with simultaneous influence of 75 dB wide band noise followed by 10 minutes rest, but did not use ear plugs.

Quaas reported that under 90 dB noise and wearing hearing protectors, subjects experienced a higher pulse rate than under control conditions; the pulse rate was higher under the hearing protected condition than under the condition of 75 dB noise without protectors. He estimated the hearing protectors may not have reduced the noise level to the inner ear below 79 dB. This study used a small group of subjects without an external control group. Seven subjects may not have been adequate to prevent the results from being confounded by multiple factors on which the individuals varied. It suggests, however, that attenuation of the noise at the ear may not effect cardiovascular response as it does hearing sensitivity.

Another quasi-experiment of Burger and Klimes (1975) also showed that while ear protectors may have protected hearing, they did not prevent the effects of noise upon the circulatory system. The researchers conveyed simulated industrial noise (noise of tank motors) to the experimental room by loud speakers and compared 20 subjects under short-term exposure (20 minutes of exercise under noise) and long-term noise exposure (two hours of exercise under acoustic pressures of 90, 100, 110 dB). Subjects were tested with and without ear protectors and showed a decrease in pulse rate with the higher noise exposure levels under both conditions but no changes in ventilation and oxygen consumption. These authors concluded that it was impossible to affirm unconditionally that exposure to noise is completely harmless. They extrapolated beyond the two hour experiment to suggest "that its negative effects will become manifest after an exposure lasting a number of years". Tests were conducted in the Latin square to avoid habituation and subjects were pretested. Information is not given to adequately judge how well the experiment actually controlled for confounding variables, especially since the groups were small and only two in number.

3.7.8 Effects of Vibration

The extent to which vibration in addition to noise may have contaminated the results of the studies on noise exposure and cardiovascular disease is unknown. Vibration was not assessed in most of the investigations. Five studies were identified in the literature search which attempted to measure vibration and noise in the worker. Although the studies attributed an increase in angina rates, in beta-lipoproteins, in the general sick rate and in disorders of the regulation of the blood pressure to vibration exposure, these studies were methodologically poor.

Rumiantsev et al (1971) reported they were able to differentiate changes related to the effect of noise and vibration from the possible effect of other environmental factors. Cholesterol, beta-lipoprotein level, blood sugar, and blood pressure of sailors employed on eight ships were measured. Criteria for determining blood values were specified; blood pressure changes were not. Comparisons were made among three groups: sailors in the engine section, sailors of the engine room given 5 mg of thiamin and 50 mg of nicotinic acid and deck crewmen who served as controls. Blood tests were conducted before a voyage and on the 15th nad 30th days of the course. Blood pressures and pulse rates were taken before and after each watch.

The authors concluded that significant increase of the sugar concentration in the blood on the 15th day of the course and beta-lipoprotein on the 30th day of the course in sailors of the engine squad could probably be treated as a consequence of noise and vibration. There were no analogous changes in blood sugar and beta-lipoproteins in the control group. Changes of the arterial pressure before and after the watch in sailors working in the engine room were likewise explained. Unfortunately, the authors failed to measure blood pressure and pulse in the control group for comparative purposes. Furthermore, these conclusions must be questioned because of multiple confounding variables and because the noise exposure groups were so poorly described.

Kangelari and colleagues (1966) studied the effect of vibration and noise on the general illness or sickness rate and reported that motor mechanics and cleaners exposed to noise had a much higher sick rate index than the control group. This was true for flu and acute common colds of the upper respiratory tract as well as diseases of the nasopharynx, breathing organs and the gastrointestinal tract. The cardiovascular response of interest, angina sick rate, was 8.9 in motor mechanics and 7.8 among the fitters or control group. When the cleaners were further divided according to those suffering from vibration disease and those not suffering from vibration illness, the angina rates were 8.7% and 5.4%, respectively. Among the cardiovascular end points, angina is by definition, very subjective. Although this study had a reasonable large group of workers, exposed to noise from 116-120 dB and unknown number of controls working in noise of 88-90 dB, it is very poorly described and offers little information for judging causal associations.

In a large study of the health of the nonspecific effects of industrial noise and vibration, Kobets and colleagues (1972) studied five groups of women: 444 employed in product warehouses and storage work requiring physical stress, 390 practically healthy women, 147 female concrete workers, 144 women who were subject to the effect of noise, and 89 persons suffering from noise disease. In their cross-sectional comparison of medical examination data and annual disability days, disorders of the regulation of arterial blood pressure were 5 to 8 times more frequent among those working in vibration and noise than those in the control group. Unfortunately, no data were presented in this study and thus, the conclusions are suspect. These authors also reported an increase in the cases of sick rate rising from the lowest in the control group to the highest rate of 3.9 times that of the controls in individuals with vibration disease. It is clear that the information reported in this study is inadequate for drawing any conclusions relative to an association between noise vibration and health outcomes.

Later Paranko and colleagues (1974) attempted to determine the effect of permissible levels of vibration in noise by studying 103 miners (61 drillers and 42 sinkers) who routinely used protection from sound and vibration. Multiple health states were considered including arterial blood pressure and heart rate, complaints, hearing loss, sexual activity, lowering of pain and vibration sensitivity, lowering of skin temperature, and increase in visual motor reaction time.

Unfortunately, the authors failed to select an appropriate comparison group, apparently choosing to evaluate their results relative to other findings in the literature rather than identifying controls. They presented no raw data on blood pressure or pulse rate. Tabled data showed an increasing proportion of workers with high blood pressure as the length of employment increased up to 16 years. Age was not controlled and selective forces were not considered. Although the authors concluded that stable functional shifts develop in miners exposed to the effect of vibration and noise at levels allowable by norms, this study is of little value because of its failure to employ an adequate control group and to specify the health and noise parameters in detail.

One of the poorer studies among the translated literature reported a tendency toward hypertension in 20 percent of the workers examined in a limestone and dolomite crushing-enriching plant and quarry of the flux-dolomite combine. In this cross-sectional study of measurements taken before and after a shift of work, Zvereva and colleagues (1975a) compared 334 workers from eight occupational groups. Health measures included arterial blood pressure, a complaints index, state of capillaries, objective symptoms such as trembling of the hand and asymmetry of arterial pressure. The quarry noise levels ranged from 95-100 dB with maximum energy in low and medium frequencies, whereas the crushing-enriching plant noise levels ranged from 86-106 dB of high frequencies. There was no evidence of a control group and no evidence of data analyzed in a before-after fashion. Even for descriptive purposes the vibration effects probably confound the noise effects in this group of workers. The conclusions lack supporting data.

3.8 Brief Summary of the Translated Literature

Forty of the 47 translated studies reviewed herein were cross-sectional in design. In the blood pressure studies, relatively small differences in systolic and diastolic pressures were observed between the high and low noise exposure groups. Compared to the English literature studies, the translated research more often included assessment of multiple confounding variables such as age, temperature, social class, etc. However, only two studies, those of Ising et al (1979) and Capellini et al (1974), applied analytic statistical techniques currently used in cardiovascular epidemiology to assess the effects of such variables as potential confounders or additional risk factors.

Similar to the findings from the English literature, the translated studies demonstrated stronger evidence of an association between noise and elevated blood pressure than other cardiovascular responses. Although the translated literature offers little data for determining risk estimates, high prevalence ratios indicating that noise exposed groups experienced higher rates of hypertension than the low noise controls were reported by Meinhart and Renker (1970), von Eiff and Neus (1980), Britanov (1979), Cieslewicz (1971) and Shatalov and Murov (1970). In addition, several cross-sectional studies provide suggestive evidence of a dose-response relationship between noise exposure and elevated blood pressure. Workers with long noise exposure in a foundry were observed to have higher blood pressures than workers with fewer years of employment, but age was only partially controlled (Folprechtova-Stenzlova and Janicek, 1966). Kalicinski et al (1975) also found that among spinners and weavers of similar ages, the frequency of hypertension was significantly greater, the longer the occupational exposure to noise. Three additional studies, judged by the review team to be less methodologically sound, reported an increase in hypertension with length of employment (Kachnyi, 1977; Sanova, 1975; Meinhart and Renker, 1970).

The translated literature offers limited data as to the influence of hearing protection on the reduction of blood pressure effects of noise exposure. Cieslewicz in 1971 and Paranko et al in 1974 concluded that individual protective devices used over the years by workers exposed to noise of 96-116 dB and greater than 85 dB respectively, did not protect them against extra-auditory effects of noise. The most promising evidence of the effects of noise diminution with ear protectors was provided in a well-designed pilot study by Ising et al (1979). Ising and colleagues reported that when working without ear protection at a mean exposure to noise of 95 dB (measured by dosimetry), the systolic blood pressure was higher by almost 7 mm Hg than when working with ear protection.

Although remarkably few investigations of the relationship of noise exposure to overt clinical disease or major cardiovascular risk factors other than blood pressure were identified in this literature review, most of them were found in the translated research. Electrocardiographic changes were investigated by twenty-one research teams, but no patterns emerged to show specific effects of noise. Changes in cholesterol under high noise exposure were reported by Rumiantsev (1971) and Khomulo et al (1967). Kanevskaia et al (1977) observed a decrease in noradrenalin in a cross-sectional group of workers while Ising et al (1979) reported an increase of noradrenalin by 16% for subjects when working without hearing protection compared to the same subjects working with hearing protection. In studies of other cardiovascular parameters, Capellini and Maroni (1974) showed the risk quota for coronary disease due to exposure to intense noise in a chemical industry to be equal that in a non-exposed population by an increase in age of 10 years; Suvorov et al (1979) observed that with each increase in the level of noise by one decibel, the neurocirculatory impairment increased by 0.5% among workers in a machine building industry; and Meinhart and Renker (1970) reported the prevalence of myocardial injuries for the noise-exposed group to be twice as high as among the normal population.

The consistency of the evidence accumulated in the translated literature, although derived primarily from cross-sectional and laboratory studies, suggests a need for systematic investigation of the relationship of noise to physiological changes and to cardiovascular disease manifestations, especially elevated blood pressure.

Section 4

SYNTHESIS OF THE LITERATURE WITH RECOMMENDATIONS

Cardiovascular disease continues to be a leading cause of death and disability in the United States. In 1972, the death rate for all cardiovascular disease was 479 per 100,000 population. The largest components of this were ischemic heart disease, with a rate of 329, and stroke with a rate of 102 per 100,000. The annual incidence rates for ischemic heart disease as manifested by Acute Myocardial Infarction in white males are estimated to be about 6 per 1000 population between the ages of 35 and 75 years. This compares with a rate of only 1.5 to 2 per thousand for white females of the same age. These figures do not include angina pectoris not progressing to infarct. Stroke, with an incidence one-third that of ischemic heart disease, still affects 400,000 people each year, 40% of whom die within a month. Approximately two-thirds of the survivors of strokes have some degree of disability. Taken together, ischemic heart disease and stroke account for 50% of the mortality in the United States.

Past epidemiologic studies have shown such variables as elevated blood pressure, elevated serum cholesterol, cigarette smoking and sedentary life style to be factors associated with increased likelihood of ischemic heart disease. Elevated blood pressure has been implicated as the cardinal risk factor for stroke. Enthusiasm over identification of several precursors of the cardiovascular diseases should not obscure the fact that these risk factors, taken together, provide an incomplete estimate of the coronary disease burden of a population and an insensitive predictor of the risk of individuals. General environmental and psychosocial and stress-related variables offer the possibility of accounting for at least part of the cause of cardiovascular disease still remaining unexplained.

While the epidemiologic evidence in favor of noise as a risk factor for cardiovascular disease is not currently strong, the case for noise involvement is so biologically plausible and the disease of such magnitude that the benefits of continued investigation using well-designed studies are desirable. The cluster of social and psychosocial stress factors which appears to play an independent role in cardiovascular disease etiology has not been clearly defined, but may indeed include noise as a principle component. Thus the role of noise in hypertension and other cardiovascular diseases, their precursors and sequella, among human populations should be elucidated and with considerable urgency.

The major objectives of this section of the report are to: (1) evaluate the overall epidemiologic evidence available on the effects of environmental noise on the cardiovascular system in man; (2) set forth recommendations for the planning of future epidemiologic studies of cardiovascular effects of noise.

This section begins with a brief description of the proposed mechanisms by which noise influences cardiovascular disease processes. It is followed by a synthesis of the major findings from the English and translated literature. A brief discussion of the major research issues culminates in a set of recommendations for future epidemiologic investigations.

4.1 Overview of Proposed Mechanisms Between Noise Exposure and Cardiovascular Disease Processes

Various theories have been postulated to suggest the mechamisms by which noise and other environmental factors contribute to the development of cardiovascular disease (as a causative, an aggravating-accelerating, or a precipitating factor). A review of all the potential mechanisms whereby noise may influence the cardiovascular system is complex and is beyond the scope of this literature review. The current status of the various theories has recently been described in some detail by Hattis and Richardson (1980). These theories provide a general framework within which to judge the plausibility of associations reported in the world literature. Since atherosclerosis and hypertension are diseases of multifactorial cause, knowledge of clearly defined risk factors is necessary in interpreting the literature which attempts to associate any factors, such as noise, as a cause. Information on general mechanisms of the pathogenesis of atherosclerotic cardiovascular disease and hypertension as well as the current knowledge on epidemiology has been reviewed in Appendix C.

Environmental noise may influence human health by direct damage to an organ or by inducing physiologic changes which may lead to adverse health effects. The physiologic changes may initiate (causative) or accelerate (aggravate or become an added risk factor) abnormal processes which if not reversed lead to clinical disease. In addition, the physiologic changes may precipitate clinical events - bring to light a cardiovascular disorder previously unknown (subclinical) or known, but tolerated (asymptomatic and controlled).

The mechanism by which these physiologic changes occur is unclear, but may be due to direct stimulation of the sensor neural system producing direct responses in the autonomic nervous and endocrine systems. In addition, noise may be perceived by higher centers as a "stress" and the body respond to this stress with specific neural and humoral responses.

Hypertension and atherosclerosis are two major underlying diseases associated with ischemic heart disease and stroke. Present epidemiologic data indicate that other environmental factors may play a role in the pathogenesis of these conditions through "stress" induced mechanisms. The present hypothesis on the pathogenesis of these conditions indicates that stimulation of the central nervous system may induce changes in peripheral vascular resistance and other cardiovascular factors inducing hypertension. In addition, hypertension itself as well as increased catecholamines, changes in other vasoactive substances, and changes in platelets and lipids may produce endothelial damage which may lead to atherosclerosis. Experimental short-term effects of noise have indicated changes in peripheral vascular resistance, elevations in blood pressure, changes in serum catecholamines and blood lipids, all of which suggest that noise acts as such a stressor, and therefore ischemic heart disease, hypertension, and stroke may be adverse health outcomes.

In addition, acute elevations in blood pressure and elevations in serum catecholamines (as well as less well defined factors) may precipitate clinical events. Increase in sympathetic nervous activity has been implicated in one clinical manifestation of ischemic heart disease - sudden death. It appears reasonable that if noise does behave as a "stress" producing acute effects, it may therefore be a risk factor in the development of hypertension and atherosclerosis. There are numerous potential cardiovascular responses to noise, only some of which have been investigated to date in study of noise as a short-term stimulus. These have been enumerated in Table 3-2 discussed on page 3-5. But, as has been pointed out by Hattis and Richardson (1980, p.6), there is currently little information on the quantitative relationships between the magnitude of short-term physiologic variations induced by stressors and the magnitudes of chronic disease processes.

4.2 Evaluating the Evidence: Criteria for Judging the Association

Epidemiologically, a causal relationship is said to exist when the evidence indicates that the factor of interest forms part of the complex of circumstances that increases the probability of occurrence of a disease and when there is evidence that a reduction in that factor decreases the frequency of the given disease.

The assessment of causality in human health is difficult at best; no single epidemiologic study or even series of studies of the same type are adequate to establish causality. At a minimum the judgment as to the strength of a causal relationship must be based upon the completeness with which the data are shown to meet the following criteria:

(1) Temporal relationships indicating that exposure precedes the disease or health response;

(2) A strong association between the factor of interest and the health outcome or disease;

(3) A dose-response relationship existing between levels of the factor and the health outcome;

(4) Interventions in human populations on the exposure which have the effect of lowering risks to the disease;

(5) Consistent findings as to type of associations and dose-response across many studies and populations;

(6) Biological plausibility of the observed associations;

(7) Findings from experimental animal studies which demonstrate the same or similar effects; and,

(8) Findings from human experiments to the extent possible, which show the effects observed in epidemiologic studies.

The goal of epidemiological research of the relationship between noise and cardiovascular disease is to establish, as clearly as possible, the evidence for criteria 1 through 5 above. Criteria 6, 7, and 8 apply to non-epidemiological research. When epidemiological observations are shown to be biologically plausible and are supported by similar findings from animal and experimental studies, the degree of certainty of the characterization of the relationship is as high as can presently be achieved.

4.3 Evaluating the Evidence: The Effect of Noise on Blood Pressure

There is general consensus that no single underlying process is responsible for increase in blood pressure, so blood pressure changes may reflect different stages in the development of hypertension or fundamentally different diseases, having in common the presence of high blood pressure. The observations that hypertension affects the rate of atherosclerosis and that cardiovascular disease risk increases with increasing blood pressure (see pp.32-38, Appendix C) make blood pressure a useful indicator of chronic pathological processes. In addition, blood pressure is among the more easily and widely measured cardiovascular parameters.

4.3.1 Temporal Relationships and Study Design

Out of a total of 83 studies reviewed herein, 55 investigated the effects of noise on blood pressure. In 44 of these 55 studies, the authors reported positive associations between noise exposure and high blood pressure or hypertension rates. The literature is strikingly similar as to the study design employed; most of the conclusions were derived from cross-sectional or prevalence data. While prevalence studies have the advantage of providing a fairly quick view of the existing illness in a population and its attributes at a given time, they can rarely establish the circumstances under which the disorder arises. The derivation of causal inferences depends, first and foremost, upon the temporal sequence between the exposure of interest (noise) and occurrence of the disease of interest (cardiovascular disease). The cross-sectional strategy, which cannot identify exposure prior to the onset of disease (or physiological changes), provides the weakest epidemiological basis from which to infer cause. The following example provided by

A. Lilienfeld (1980, pp.194-195) clarifies the time distinction between cross-sectional and retrospective studies:

> To illustrate the difference in inferences that can be derived from cross-sectional and retrospective studies, assume that one is interested in the relationship between cerebrovascular disease (stroke) and the level of serum cholesterol A group of stroke cases and appropriate controls are selected, and blood is drawn to determine their serum cholesterol levels. If the levels are significantly higher among the cases than the controls, a statistical association is said to exist between stroke and elevated cholesterol levels. In this cross-sectional approach, however, one does not know whether the elevated serum cholesterol preceded the onset of the stroke or followed it. If the latter were true, obviously the elevated serum cholesterol could not be regarded as being etiologically important in the development of stroke. On the other hand, in a retrospective study, one would seek information on the level of serum cholesterol that was present before the onset of the stroke. If a statistical association is then established, a causal relationship can be inferred with greater confidence.

Cohort studies or carefully contrived "incident" case-control studies are needed to clearly distinguish antecedent from consequence. Needless to say, poorly designed cross-sectional studies contribute even less to sound conclusions. A good prevalence study begins with a representative sample of one definable population. Yet, few of the researchers conducting the studies reviewed herein described the populations at risk, details of the sampling frame or completeness of the sample.

Potential selection biases inherent in cross-sectional data, such as selective survival may distort the observed association in either direction. The problem of selective survival is that those persons who develop the disease but who die early will not be counted in the study population. In any situation where disease, disability, or employment practices differentially impact on the noise and non-noise exposed individuals, prevalence data for the two groups may be distorted. Selection bias does not necessarily invalidate study findings but should be evaluated for any given investigation. In the majority of the studies, potential for selection bias existed with no exploration of the problem by the author(s).

Since 66 of the total 83 research papers reviewed herein, described study designs of a cross-sectional nature, it is not possible to conclude that noise exposure precedes an increase in blood pressure or onset of other cardiovascular diseases. Four studies investigating blood pressure effects of noise employed designs of a prospective nature, but unfortunately suffered from major methodological shortcomings. In the S. Cohen et al (1981c) prospective study, subjects from the noisy areas with the higher blood pressures on initial examination were lost to follow-up. Brown et al's (1975) historical prospective analysis showed no changes in blood pressure due to noise exposure, but failed to account for the total cohort of workers and failed to control for confounding variables. Friedlander and colleagues (undated) reported a trend toward elevated blood pressure with noise exposure from a historical prospective pilot study, but the sample was highly selective and extant medical record data were used without verification. Ising et al (1979) demonstrated positive effects of noise on blood pressure in an intervention study of workers using hearing protection. However, the intervention was short-term with hearing protection effects studied for one week only. Their cross-sectional data revealed no differences in blood pressure between high and low noise groups. Clearly, if the temporal relationships between noise exposure and health outcomes are to be elucidated, studies of a prospective nature must be conducted.

4.3.2 Strength of the Association

A strong association and gradients of risk from low to high levels reduce the likelihood that an observed association is spurious. Our review of the published literature reveals a paucity of information from which to quantify effects. An attempt was made to systematically quantify the magnitude of the differences reported in systolic blood pressure between the high and low noise exposure groups. Twenty studies, five of them quasi-experimental, were identified which provided mean systolic blood pressure values and data on exposure. Figure 4-1 indicates that relatively small differences in systolic blood pressure have been detected. When one considers that blood pressure measurements are sensitive to a variety of outside stimuli such as temperature and level of hydration, small differences derived from cross-sectional data are difficult to interpret. Interpretation is especially difficult when the possibility of selection bias and measurement error exist. Such small differences in mean systolic blood pressures observed cross-sectionally without consideration of age and sex effects, may also be of doubtful biological significance.

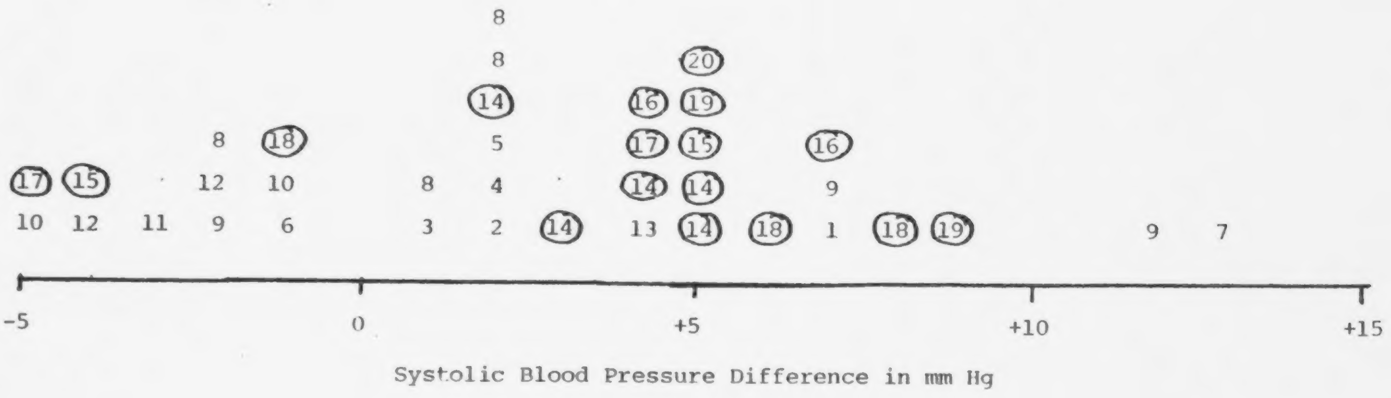


Figure 4-1. Reported difference in systolic blood pressure between high and low noise exposure groups*. Multiple results are shown.

*Circled numbers indicate translated literature and uncircled numbers indicate English language literature.

1. Antonova, K.P. (1971)
2. Brown, J.E., et al (1975)
3. Cohen, A., et al (1980a)
4. Cohen, S., et al (1980b)
5. Cohen, S., et al (1981c)
6. Hedstrand, H., et al (1977)
7. Jonsson, A., et al (1977)
8. Lees, R.E.M., et al (1979)
9. Manninen, O., et al (1979)
10. Mosskov, J.I., et al (1977a)
11. Mosskov, J.I., et al (1977b)
12. Mosskov, J.I., et al (1977c)
13. Takala, J., et al (1977)
14. Andrukovich, A.I. (1965)
15. Folprechtova-Stenzlova, A., et al (1966)
16. Ising, H., et al (1979)
17. Pokrovskii, N.N. (1966)
18. Sanova, A.G. (1975)
19. Shatalov, N.N., et al (1969c)
20. Tr[illegible]anskii, M.P., et al (1971)

No notable study nor set of studies was identified which would allow a reasonable quantitative estimate of the relative importance of noise in producing cardiovascular disease and hypertensive effects. The estimation of the effect of a given factor in producing disease, referred to as the relative risk, is usually obtained directly in cohort studies, or indirectly from the odds ratio in case control studies. Because of the proponderance of cross-sectional data it is very difficult to estimate the risk that noise exposure might contribute to increases in blood pressure. Friedlander et al (undated) reported 35-44 year old men in the high noise group to have 2.76 times the risk of developing increased diastolic blood pressure and 6.4 times the risk of developing high systolic blood pressure of similar aged men in the low noise group. This difference in risk, however, cannot be ascribed to noise exposure only; there were many other differences between the high and low noise groups, resulting in the strong possibility that the difference may be due to selection and other biasing factors.

The research available for review of the influence of noise on blood pressure is notable in its poor handling of potentially confounding variables. In a multi-causal model of disease causation, one expects several characteristics to contribute to the onset of the health problem. Potential confounders or control variables are exposure characteristics other than the hypothetical causal factors (noise) which are associated with the disease of interest (cardiovascular responses) and with the hypothetical causal factors (noise). Confounding occurs only in the context of a given study; the extraneous variable or confounder must be associated with (noise) exposure in the data; thus, the same variable which confounds in one study may not confound the same association in another. Confounding occurs when presence of the extraneous factor distorts the estimation of the effect of the factor (noise) in producing disease. It is clear from the summarized data in Appendix A that few extraneous risk factors were controlled in the research on noise to date. Age and sex, strong risk indicators in cardiovascular disease which have also been shown to be associated with noise exposure were the most frequently considered factors and were often not adequately controlled in the statistical analysis.

Ten of the studies of blood pressure in the literature evaluated reported collecting data on multiple potential confounders and covariables such as smoking, diet, exercise, fluid loss, hearing loss, etc. Only three of these groups of researchers, Ising et al (1979), Capellini et al (1974) and S. Cohen et al (1981c), applied analytic statistical techniques currently used in cardiovascular epidemiology. Overall, there was little evidence of an attempt to study concomitantly with noise, other factors which may have been contributing to the causal network.

Although the reviewed literature offers little data for determining risk estimates, several studies lend some qualitative support to the hypothesized relationship that noise adversely affects blood pressure. High prevalence ratios indicating that the noise exposed group experienced higher rates of hypertension than the low noise controls were reported by Meinhart and Renker (1970), von Eiff and Neus (1980), Britanov (1979), Cieslewicz (1971) and Shatalov and Murov (1970). Meinhart et al (1970) reported prevalence ratios of hypertension between noise impaired and controls for 12-40 year olds of 7.6, for 40-65 year olds, 9.7 and for 65 and older individuals, 8.2. Von Eiff et al (1980) noted that 22.8% of the high noise area residents indicated existing hypertension compared to 14.6% of the low noise area residents in his community survey. Britanov (1979) reported a prevalence ratio of 2.8 and Cieslewicz (1971) showed prevalence ratios of hypertension between weavers and spinners of 2.2 for women and 2.7 for men. Shatalov and Murov (1970d) demonstrated age-sex adjusted prevalence rates of hypertension among men exposed to the combined effects of tension and noise to be 13.57 compared to 3.95 for the controls. While these data are suggestive of an association between noise exposure and hypertension, they must not be taken to represent risk estimates since they were not derived from incidence data. Furthermore, these prevalence ratios are based on data from studies that were judged by expert reviewers on this project to be of relatively poor scientific quality. (See Tables 3-6 and 3-7 for ratings.)

In summary, although the literature provides weak quantitative evidence of an association between high noise level and adverse blood pressure effects, the trends in the data and the consistently high prevalence ratios indicate further investigation is warranted.

4.3.3 Dose-Response Relationship

It would be relevant and very useful to present a summary of the blood pressure changes associated with degree of change in noise exposure, but this necessarily requires consistency among studies in the specification of noise exposure levels and blood pressure values which this body of literature lacks. However, several studies provide suggestive evidence of a dose-response relationship between noise exposure and blood pressure changes. S. Cohen et al (1980b) demonstrated that blood pressure in children increases with years exposed, but age effects were not considered in the cross-sectional data. Knipschild (1977a) observed higher blood pressures as the level of airplane noise increased in the villages; Parvizpoor (1976) demonstrated an increase in the prevalence of hypertension with increase in length of employment as a weaver; Kachnyi (1977) found an increase in hypertensives with length of employment among weavers working in high noise, but made no comparisons with control subjects; Sanova (1975) observed an increase in the hypertension rate among noise exposed compressor operators, but failed to make comparisons with his control subjects. Meinhart and Renker (1970) reported that hypertension began rising after five years employment and rose precipitously after 20 years of work.

Two additional studies, judged by the review team to be among the better translated research, provided data indicating possible dose-response relationships. Workers with a longer noise exposure (determined by years of employment in a foundry) were observed to have higher blood pressures than workers with fewer years of employment, but age was only partially controlled (Folprechtova-Stenzlova and Janicek, 1966). In a study of spinners and weavers, the frequency of hypertension was significantly greater, the longer the occupational exposure to noise: 26% of the women working 1-6 years in noise compared to 38% working 7-12 years and 47% working 13 or more years in noise were reported to be hypertensive (Kalincinski et al, 1975). The mean ages of the groups were similar.

Use of years of employment in a noisy environment as a surrogate exposure variable for correlation with blood pressure to estimate dose-response must be treated as presumptive evidence even when analyzed appropriately and interpreted cautiously. Since age increases with years employed (duration of exposure) and blood pressure increases with age, it is essential that there be appropriate control for the potential confounding of age. None of the eight studies suggesting the possibility of a gradient of response adequately controlled for age effects.

Although an observed dose-response relationship makes a causal hypothesis more plausible, it is sometimes impossible in epidemiological studies to obtain quantitative estimates of the degree of exposure to a potential etiological agent. In the case of the effects of noise on blood pressure, the possibility of obtaining quantitative estimates by degree of exposure has not been adequately exploited. The observed associations with years of employment would suggest that further exploration is in order. In fact, reanalysis of existing data such as that published by Kalicinski et al (1975) with reconstruction of the noise exposure "cohorts" and adequate controlling for age and other potential confounding variables may offer more definitive evidence relative to the effects of long-term noise exposure on blood pressure.

4.3.4 Effects of Intervention on Noise Exposure

A reduction of the increase in blood pressure with use of hearing protectors or other noise abatement measures in high noise environments would offer strong support for the hypothesis that noise adversely affects cardiovascular health given that noise effects are mediated via the sensorineural systems. The literature offers little and conflicting evidence as to the influence of hearing protection on the reduction of blood pressure effects of noise. Variation in the actual noise level reductions provided by given devices, worker noncompliance and ethical problems in assigning "no use" of hearing protection under high noise levels makes study in this area difficult.

The pilot study by Ising et al (1979) in which 12 subjects wore hearing protectors during work for one week and worked without hearing protection for one week of the study period provides the strongest evidence. The workers wore dosimeters and the actual daily noise level reduction provided by the hearing protection was determined to be between 10-16 dB(A) when averaged over the entire day. Ising and colleagues reported that when working without ear protection at a mean exposure to noise of 95 dB(A), the systolic blood pressure was higher by almost 7 mm Hg than when working with ear protection.

S. Cohen et al (1981c) reported no significant differences of blood pressure for noise exposed children who had experienced a year in noise-abated classrooms compared to the continuously noise exposed children. However, sample size was small and conceptual problems in handling the blood pressure change scores when initial values were significantly different, resulted in failure to make comparisons with the quiet-classroom children.

Cieslewicz in 1971 and Paranko and colleagues in 1974 concluded that individual protective devices used over the years by workers exposed to noise of 96-116 dB and greater than 85 dB respectively, did not protect them against extra-auditory effects of noise. Both of these investigators derived their conclusions from cross-sectional analyses of workers routinely using hearing protection as it was introduced into the work place.

With the exception of Ising and colleagues, the degree of noise attenuation of the ear protectors and the compliance in use of the protectors was not verified. Careful study of blood pressure effects with diminution of noise through constant and appropriate use of ear protectors may be one of the more promising means for further elucidating the relationship of noise to health effects, especially among industrial populations.

4.3.5 Consistency of Findings

Confirmation by repeated findings of an association in different population groups and different countries strengthens an inference of a causal connection. Data derived from retrospective and prospective studies offer the strongest confirmatory evidence. As indicated previously, 44 of the 55 blood pressure studies reported adverse associations between noise exposure and blood pressure. Although cross-sectional designs predominated, these observations suggest that deleterious health effects may result from exposure to high noise levels. Unfortunately, several of the more scientifically rigorous studies reviewed herein which might have confirmed some of the cross-sectional observations suffer from small sample size and other design problems. Seven such studies reported no adverse effects of noise on blood pressure. Brown et al (1975), in a historical prospective design employing 29 pilots and 29 non-flying executives as controls, found that changes in blood pressure over a seven year period were not statistically significantly different between the pilot and control group. No significant differences were observed for new cases of hypertension over a 15 year period for the 70 matched-pairs of production and material handling workers studied by

Lees, Smith and Wetherall (1980). Likewise, Capellini and Maroni (1974) found no associations between noise and hypertension although their data indicated an increased risk quota for coronary disease due to exposure to intense noise. Drettner (1975), Takala et al (1977), and Hedstrand et al (1977) reported no significant associations between noise exposure or noise-induced hearing loss and blood pressure. Ising et al (1979) in their seminal study of noise effects on brewery workers observed intra-individual differences in blood pressure in the effect of the use of ear protectors, but failed to observe statistically significant differences in systolic and diastolic blood pressures between the noise exposed and control groups. However, the differences reported were suggestive of a noise effect and the failure to reach statistical significance may be related to small sample size. Likewise small sample size may account for the failure to observe differences between the noise exposed groups in the studies by Brown et al (1975), Lees et al (1980), and Takala et al (1977).

Part of the discrepancy in findings from studies of noise and health effects may arise from differing degrees of reliability and validity of the measurements. The literature is remarkably consistent in its failure to consider the validity and reliability of both exposure and outcome measures and the effect of misclassification errors on observed associations and statistical procedures.

Several ways to improve measurement are to: (1) apply clear and reproducible criteria in defining and classifying exposure and outcome status; (2) apply criteria which provide manifestationally homogeneous groups; (3) validate subjective data with objective measures when subjective data are useful; (4) use objective tests with known validity or accuracy rather than subjective measures whenever possible; (5) assess both exposure and outcome variables using multiple tests; (6) obtain information in such a manner that subjects are unaware of the objectives of the investigation and observers are unaware of the conditions on which subjects vary; (7) maintain quality control over the measurement procedure; (8) assemble data on the exposed and non-exposed subjects in a comparable manner; (9) keep the non-response low and compare respondents and non-respondents with respect to ancillary information; (10) take several assessments of exposure and outcome measures to judge change over time; and (11) to assure comparability of reporting, compare groups of interest on the frequency of reporting experiences which seem unlikely to be relevant to the etiology under investigation.

The major problem in assessing noise exposure was the lack of information provided by the researchers. The major omissions were frequency composition, duration, instrumentation and measurement procedures, subject characteristics and background noise level. With the exception of Ising et al (1979), no study derived individual noise exposure data based on dosimeter measurement. Of the six population-based studies reported in the English literature with our highest noise exposure ratings, not one provided adequate data to permit the determination of noise dose. In several papers there was no evidence of any measurement of noise exposure for the control group. The most frequently reported noise descriptor was the noise level given in dB with no weighting specified or in A weighted dB. In the translated literature, four of the eight studies receiving our highest noise exposure ratings were experimental in design; the four studies conducted in occupational settings described the noise exposure in reasonable detail and indicated that standardized equipment and measuring procedures were used. The latter four studies were those of Pilawska et al (1977), Folprechtova-Stenzlova et al (1966), Britanov (1979) and Sanova (1975). In the translated literature, the actual noise levels studied were consistently higher than the levels reported in the English literature, especially the United States literature. Very few "low noise", that is, groups exposed continuously to less than 75 dB noise were included in the investigations. Thus, although a range of noise levels was reported, the noise levels studied were remarkably high.

In general, the quality control of blood pressure measurement was less than adequate. Although a few studies were very precise in defining blood pressure measurements and reported that observers were unaware of the noise exposure status of the subjects, most studies provided little information. The hypertension data are difficult to compare because various blood pressure levels were used to define hypertensive states. Summary tables A-1 through A-10 in Appendix A show the range of definitions of hypertension employed. Improvements in the measurement of exposure and health outcome variables, as well as study design, would appear necessary for the confirmation of the associations observed in the large body of cross-sectional studies.

4.3.6 Summary of the Evidence of the Effect of Noise on Blood Pressure

This review group concludes that there is sufficient evidence in the existing world literature to make further investigation necessary into the question as to whether continuous exposure to high noise is associated with increased blood pressure, especially among industrial workers. Careful analysis of the available research relating noise exposures to changes in blood pressure reveals that cross-sectional studies have repeatedly demonstrated that blood pressure is higher among individuals continuously exposed to high as opposed to low levels of noise; prevalence ratios for blood pressure between high and low noise exposure groups are consistently high; and findings from epidemiological studies are consistent with those from other research methods. In addition, several studies provide presumptive evidence of a dose-response relationship and a recent, well-designed, pilot study suggests that noise attenuation with ear protectors may reduce the impact of noise on blood pressure among industrial workers.

Several limitations of the evidence at present are that the above observations are derived primarily from cross-sectional data from which it is impossible to determine that noise exposure precedes the cardiovascular response; the data showing adequate controlling for possible confounding variables are not available to determine that the observed associations are independent; the more advanced and powerful analytic techniques currently applied in cardiovascular epidemiology have not been employed. Although cross-sectional data are inadequate for quantitative analysis of risk and for generating dose-response curves, they provide excellent sets of reference data. This accumulation of cross-sectional studies and their continued use in the study of the effects of noise on the cardiovascular system can be likened to the time required for the evolution of appropriate study designs to rigorously test hypotheses and present evidence in favor of cigarette smoking being a risk factor for lung cancer, coronary heart disease and other illnesses.

The importance of these observations, although primarily from cross-sectional studies, becomes apparent when one considers that elevated blood pressure is recognized as the cardinal risk factor for stroke and one of the three prime risk factors for ischemic heart disease. In the United States alone, these two diseases account for 50% of the mortality. Environmental and psychosocial stress factors which appear to play an independent role in cardiovascular disease, may indeed include noise as a principle component.

While the epidemiologic evidence in favor of noise as a risk factor for cardiovascular disease, especially hypertension, is not currently strong, the case for noise involvement is so biologically plausible and the disease of such magnitude that the benefits of continued investigation using well designed studies are desirable. Thus the role of noise in hypertension, its precursors, and sequellae, among human populations should be elucidated and with considerable urgency.

4.4 Evaluating the Evidence: The Effect of Noise on Cardiovascular Parameters Other than Blood Pressure

A major concern in the assessment of health effects is the very limited range of physiological parameters, other than blood pressure, such as lipid levels and pathologic end points which have been studied. Only three studies used "incident" cases of overt cardiovascular disease, Hannunkari (1978), Raytheon (1975), Lees, Smith and Wetherall (1980) and without exception, failed to specify diagnostic criteria applied.

Meinhart and Renker (1970) reported prevalence of myocardial injuries for the noise-injured group to be twice as high as among the normal population, but provided no diagnostic criteria. Likewise, without stating diagnostic criteria, Tavtin (1976) and Kobets et al (1972) reported functional disorders of heart action and disability to be higher among the noise exposed than non-exposed; a higher frequency of complaints including angina was noted by Koszarny et al (1976), Britanov (1979) and Kangelari et al (1966).

Electrocardiographic changes were investigated by twenty-two research teams with bradycardia or tachycardia most frequently reported, but no pattern emerged to show specific effects of noise. Pokrovskii (1966) reported bradycardia or tachycardia occurring on the average five times higher for persons exposed to intense noise than for the controls. Cuesdean et al (1977) studied ST depression, Yazburskis (1971) measured ST changes of the "ischemic type", and Kalicinski et al (1975) found no differences between subject groups as to ST changes indicating inadequate blood supply to the heart. Several of the electrocardiogram alterations were judged by the cardiologist reviewing the literature herein to be normal variants without clinical evidence of associated disease. However, findings were interpreted by the authors to be indicative of parasympathetic system dominance. Other parameters found to be associated with noise exposure for which we have no knowledge of a relationship to disease were heart minute volume according to Starr's formula reported by Dega and Klajman (1977), contractile blood volume reported by Sanova (1975) and minute blood volumes reported by Gel'tishcheva (1980).

Several researchers have studied parameters which are more biologically plausible relative to assessing the impact of noise on the cardiovascular system. Three research groups reported changes in cholesterol under high noise exposures. Rumiantsev (1971) observed a significant increase in beta-lipoproteins in blood serum of noise exposed sailors on the 30th day of their course, and di Cantogno et al (1976) reported that noise had an effect on total lipids and triglycerides. Ising et al (1979) studied cholesterol but failed to report any changes if they were observed. Khomulo et al (1967) in a study of 103 workers exposed to 117 dB noise and 51 controls, demonstrated a change in cholesterol values with the increase in employment duration among individuals exposed to noise suggestive of a dose-response relationship.

Several investigators have studied the effect of noise on the sympatho-adrenalin system. Kanevskaia et al (1977) reported a decrease in noradrenalin in a cross-sectional group of workers while Ising et al (1979) reported an increase by 16% for subjects when working without hearing protection compared to the same subjects working with hearing protection.

Several additional studies provide suggestive evidence that noise adversely affects various parameters of the cardiovascular system. Dega et al (1977) showed a decrease in heart minute volume to almost the level of the control group when 10 propeller grinder operators were examined while using ear protectors; Knipschild (1977b) found that the contact rate for cardiovascular problems increased with increasing noise level for 15-64 year olds; Suvorov et al (1979) observed that with each increase in the level of noise by one dBA, the neurocirculatory impairment increased by 0.5% among workers in a machine building industry; Capellini and Maroni (1974) showed the risk quota for coronary disease due to exposure to intense noise in a chemical industry to be equal that in a non-exposed population by an increase in age of 10 years; the Raytheon study (1975 and A. Cohen, 1976) reported that workers judged to have always used ear protectors showed the greatest reduction in medical problems while those who never used the protectors showed the smallest changes. Two groups of investigators, Burger et al (1975) and Quaas et al (1970), using healthy young males in experimental conditions, concluded that the use of hearing protectors did not prevent the influence of noise on pulse rate.

4.4.1 Summary of the Evidence of the Effect of Noise on Cardiovascular Parameters Other than Blood Pressure

The review of the literature indicates there have been remarkably few investigations exploring the relationship of noise exposure to overt clinical disease or major cardiovascular risk factors other than blood pressure. The evidence of general cardiovascular adverse effects, other than blood pressure, of noise is weak and fragmentary. These cross-sectional and short-term laboratory experiments indicate the need for systematic study of the relationship of noise to physiological changes along the biological gradient of disease and to cardiovascular disease manifestations.

4.5 Discussion of Issues Related to Noise Effects Epidemiology with Recommendations

Careful analysis of the literature relating cardiovascular response to noise exposure indicates that the studies to date are less than fully informative for establishing cause-effect relationships. In the opinion of this review team, the strongest evidence of an association, if one exists, is between exposure to high noise levels and elevated blood pressure. Furthermore, it is concluded that the body of data, primarily cross-sectional in nature, provides sufficient evidence to support further research of the effects of noise on the human cardiovascular system. The need for additional knowledge of the potential impact of noise on human health is highlighted by the fact that millions of people are exposed to multiple sources and varying levels of noise in our environment. It is estimated that some 10.5 millions of people are exposed annually to noise at levels of 80 dB or greater from occupational sources; approximately 16.2 million are similarly exposed through non-occupational activities; and even larger numbers of people are exposed to noise levels of less than 80 dB from multiple sources in our society such as traffic and aircraft.

Of the 83 studies critically reviewed by the technical team composed of an audiologist, cardiologist and cardiovascular disease epidemiologist, 66 were cross-sectional in design. As indicated in Table 4-1, this is one of the weaker research designs for deriving data from which to infer causality, primarily because the temporal relationships of the onset of the factors under investigation are unclear. At best, cross-sectional data serve to describe in detail the dimensions of the problem in need of study and to generate hypotheses for testing in more rigorously designed research.

Analysis revealed that deficiencies in at least four major aspects of study design exist in the noise effects epidemiologic literature to date. Deficits were identified in the areas of exposure characterization, outcome specification, sample selection and data analysis. Weaknesses in any one of these areas lead to substantial problems in interpretation of study findings, making it difficult, if not impossible, to derive valid conclusions from the data. The following section presents a discussion of issues and recommendations focusing on the weaknesses observed in these four major areas of study design.

4.6 Exposure Characterization

In our survey of the cardiovascular-noise research, difficulties in assessing the potential causal factor of interest, noise, appeared as a major impediment to the systematic exploration of the association between noise levels and disease manifestation and to the replication of studies within and among countries. Four major gaps in the development of the exposure variable which are critical to epidemiologic research are immediately apparent: (1) lack of a common noise exposure descriptor which is reliable, valid and can be applied uniformly across situations; (2) inability to document direct exposures to the individual; (3) failure to describe the total cumulative long-term exposures taking into account non-occupational as well as occupational exposures; and (4) use of a widely varying and overlapping range of noise levels, which restricts dose-response analyses.

Table 4-1

Summary of Methodological Considerations

	Design			
Methodological Attributes	Randomized Intervention Trial	Cohort	Case-Control	Cross-Sectional
Derivation of Causal Inference				
Temporal Sequence	+++	+++	++	+
Longitudinal Measure of Exposure	+++	+++	++	+
Study of Spectrum of Morbidity Longitudinally	+++	+++	+	+
Risk Estimates				
Relative Risk	+++	+++	++[1]	
Attributable Risk	+	+++	+	++
Odds Ratio	+++	++	+++	+
Avoidance of Confounding Bias	+++	++	++	+
Avoidance of Selection Bias	++	++	+	++
Avoidance of Measurement Bias	+++	+++	+	++
Dose-Response	+++	+++	++	++
Time Efficiency	+	++[2]	++	++[3]
Cost Efficiency	+	++[2]	+++	++[3]

Key: +++ : strong design
++ : fairly strong design
+ : weak design

Definitions:

Cohort may also be referred to as prospective, historical prospective, retrospective cohort, longitudinal, indicating individuals are followed over time identifying exposures prior to incidence of disease.

Case-Control may be referred to as retrospective indicating the tracing of exposure backwards in time after disease is manifest.

Cross-Sectional may also be referred to as prevalence studies.

Footnotes:

1. The odds ratio estimated from retrospective studies is a good estimate of relative risk when prevalence of disease is low.
2. Efficiency of cohort studies is dependent upon whether exposure of the cohort can be assessed and described retrospectively or whether the cohort must be followed from the present for a period of years.
3. Cross-sectional analysis of extant data is more efficient than the above rating indicates.
4. Blank indicates not applicable.

4.6.1 Lack of a Common Noise Descriptor

Synthesis of the findings from the world literature was severely restricted by the lack of common noise descriptors and the failure to use uniform, reliable and valid methods of measurements. This deterrent to comparison and replication of research has been recognized repeatedly, but apparently has had little impact on recent nonauditory noise research. At the 1973 International Congress on Noise as a Public Health Problem, Pearsons (1973) identified six possible ways for direct measurement of noise, two graphical measures, nine measures to calculate the noise of individual events and thirteen measures to evaluate the severeness of exposure of communities to multiple events.

After the 1973 conference, acknowledging the complexity of deriving a simple indicator of noise exposure for health (primarily hearing loss and annoyance) studies, the U.S. Environmental Protection Agency selected the long-term average sound level, L_{eq}, A-weighted scale, as the best descriptor for specifying the magnitude of the environmental noise. This relatively simple descriptor can be used when the effects under consideration span an 8-hour work period, a 24-hour work period or a year period. In addition to monitoring noise over a long period of time, the L_{eq} is easily measured with equipment available and correlates well with known effects of noise on hearing, speech and general well-being. A major disadvantage of L_{eq} as an overall descriptor is its inadequacy for assessing impulse and high level noise because of the clipping of the peaks by 15-30 dB. Since much occupational noise is impulse noise more elaborate descriptors or modifications which take into account impulse noise superimposed on background noise may be the most appropriate models for future research. Nevertheless, general agreement (by scientists worldwide) on a basic descriptor such as the L_{eq} to characterize noise exposure would make it possible to describe noise environments and their change in time and subsequently the changing exposures of individuals and populations.

Our analysis revealed that of the thirty investigations published in the English literature since 1973, twenty-six reported sound level in decibel units, using the A weighted scale (see Summary Tables, Appendix A). The equivalent sound level (L_{eq}) tended to be used in transportation noise studies without specifying the time intervals over which the levels were evaluated.

The summary tables also show that five of the six studies employing hearing loss as a surrogate measure have been reported since the Dubrovnik, Yugoslavia conference.

The reviewed literature consistently failed to document reliability and validity of the noise measurements taken and provided insufficient detail for replication of the studies. The variety of methods and approaches to noise assessment appearing in the literature to date hinders replication and comparison of findings, especially among countries. The brevity of the noise descriptions and perhaps the loss of meaning in translation make it difficult to determine comparability of research even at the gross level of characterization of noise over time versus an average noise level determination. Standardized measures of the exposure variable would greatly increase the opportunity to observe consistencies of associations among studies which could, in turn, be considered logically equivalent to the replication of results in laboratory experiments under a variety of experimental conditions. Consistencies of association would indirectly strengthen the inference of an association between noise and cardiovascular responses.

4.6.2 Inability to Document Individual Exposures

Technical advances in electronics during the past decade have made it possible to assess continuous noise exposure of individuals with personally worn dosimeters. Von Gierke and colleagues (von Gierke et al, 1980; Fairman and Johnson, 1979; Johnson and Farina, 1977) have demonstrated the feasibility of the use of dosimeters by individuals over fairly long periods of time, identifying the major noise sources contributing to the total noise dose by brief diary entries. The assessment of noise by dosimeter has been or is currently being explored for use in the general urban population (Schori, 1978), with children (Roche et al, 1978 and 1979), in military populations (Fairman and Johnson, 1979) and in special groups such as housewives and factory workers (von Gierke, 1974).

Clearly, use of advanced techniques in noise measurement would greatly enhance scientific investigations since observed associations which are most likely to be biologically significant are those derived from direct measure of noise exposure to individuals who subsequently manifest disease states. The study by Ising and colleagues in 1979 was the only investigation in the natural work environment found which documented individual exposures with dosimetry readings. Among the better measures described in the literature were area noise level assessments such as those described by Pilawska et al (1977) and Dega and Klajman (1977) in studies of shipyard workers, and job title estimates of noise levels used by the Raytheon Service Company study (1975) and Brown et al (1975). Six English studies and one translated study selected hearing loss as a surrogate measure for noise exposure. The scientific contribution of these studies is limited because of the unknown relationship between hearing impairment and blood pressure.

4.6.3 Failure to Describe Cumulative Long-Term Exposures

Another major weakness in the characterization of the exposure variable is the inability to estimate total noise exposures including non-occupational as well as occupational exposures and related durations. Von Gierke et al (1980) and Johnson and Farina (1977) have demonstrated that for short-term measurements, individuals may show single-day dosimeter readings in which their off-work noise exposures exceed their work exposure levels. However, if monitored long enough, the at-work average exposures turn out to be higher than the average non-occupational exposures as has been assumed in much of the epidemiologic literature. Ising et al (1979) reported sharp differences between individual subjects when they attempted to measure noise levels of Sunday activities and were unable to use the Sunday noise levels as control values for their subjects. Nonetheless, von Gierke and others (1980) consider the evidence of non-occupational noise exposure to be impressive enough to warrant consideration in future studies of the potential non-auditory health effects of noise. It is likely that non-occupational exposures have been underestimated in the past and their contribution to the development of health problems in high risk groups have yet to be explored.

The effects of total noise exposure, whether occupational or non-occupational or both, cannot be adequately evaluated unless some assessment of duration is included in the noise model. A model which incorporates average duration of noise exposure expressed in long-term units such as years and/or months would be desirable for epidemiologic studies of cardiovascular diseases. In the reviewed literature, most of the studies expressed exposure in the very simplest of models - a general description of the noise level in the work site, measured one or more times with no linkage to the worker going through the site in the normal work day. Years of employment in the given industry were treated as a surrogate measure for duration of exposure by several investigators (Folprechtova-Stenzlova et al, 1966; Suvorov et al, 1979; Andriukin, 1961; Manninen and Aro, 1979; and Parvizpoor, 1976).

4.6.4 Restriction of Dose-Response Analyses

If noise is of causal importance in cardiovascular disease, then the risk of developing the disease should be related to degree of exposure to noise. The literature to date offers little other than presumptive evidence for or against a dose-response relationship. A major gap in the research is the lack of a range of noise levels explored. Low noise levels appearing in the translated literature are often levels considered to be high in the United States. This is especially true in recent years with the current occupational noise standards for industry set at a maximum of 90 dB for eight hours. Clearly, there is a need to develop future research utilizing a range of accepted "levels" for categorizing exposure to facilitate the search for dose-response relationships within given studies and, perhaps in an ecological sense, across studies.

Although this review reports as presumptive evidence of a dose-response relationship, an increase in blood pressure with an increasing number of years employed, this interpretation must be accepted with caution because of the potential confounding of age. Blood pressure increases with age. Age increases with years employed, thus age must be appropriately controlled for such an interpretation to be valid.

Most striking is the observation of the variability and overlap in noise levels identified, making it virtually impossible to group studies by the levels and/or range of exposure explored by the investigators. For example, Knipschild (1977a, 1977b, 1979) in a series of studies of the same general population apparently found it difficult to classify the exposure areas with uniformity across a drug study, general practice and community survey.

4.7 Recommendations Regarding Noise Exposure Characterization

Based on our analysis of the world literature and consideration of the technology available to date, we offer the following recommendations regarding noise exposure characterization for epidemiologic purposes:

(1) Researchers and writers should strive toward common agreement on the use of noise-related terms. Utilization of different terms to describe the same phenomenon only encourages confusion. ANSI standards should be used as the recognized guides for terminology. International acceptance of standardized terms is necessary if world literature on noise is to be utilized to advantage.

(2) If noise studies are to be useful, understood, and have the capability of being replicated, adequate information must be given regarding important elements such as the acoustical nature of the noise, its source, why, how and with what instrumentation it was measured, where it was measured, the actual noise levels and durations involved in the exposures, and information on subjects' hearing and physical health and their history of avocational as well as vocational noise exposure.

  (a) Descriptions of noise should include type (as steady, nonsteady), frequency composition, actual levels, and durations to facilitate dose-response determinations.

  (b) Dosimeters should be utilized vocationally and avocationally to insure accurate assessments of noise exposure. Further, dosimeters should be refined to measure external ear canal levels by utilizing a miniature ear canal microphone as well as a body-worn microphone in dosimeter design.

  (c) Researchers should refer to standards on noise measurement, such as ANSI S1.13-1971(R1976), to assure conformity to acceptable measurement methods, and to standards on instrumentation, such as ANSI S1.4-1971(R1976), for appropriate selection of instrumentation according to the accuracy needed for a particular measurement.

  (d) Calibration of instrumentation prior to, during, and following noise measurement should be accomplished and documented to assure accuracy.

(e) Studies should include environmental maps or diagrams which relate the noise in question to the ambient noise levels and the locations of persons located in the noise environment.

(f) Histories of noise exposure vocationally and avocationally should be extensive and complete for each subject in studies of noise exposure.

(g) Hearing thresholds should be established audiometrically for each subject prior to and following noise studies so that hearing function can be realistically assessed relative to the non-auditory information being studied.

(h) Physical examinations should include thorough otological examinations in any noise study.

(3) Acceptance of general "levels" for categorizing exposures which will allow comparison among studies and replicability of studies. Proposed levels are as follows:

high level > 85 dB (for eight-hour period)
medium level = 70 dB
low level < 70 dB

(4) Development of several noise characterization paradigms which would be appropriate for future studies given the type of study design and level of noise measurement possible.

Example: Noise Exposure Paradigm I (Gamble and Spirtas, 1976)

(a) Select industries (or noise areas within an industry) which vary markedly by noise levels, but are similar on worker and environmental characteristics;

(b) Have industrial hygienist and an expert in acoustics, rank and classify each job for noise exposure on the basis of assessments of the noise levels at the worksite during times workers are present;

(c) If jobs cannot be classified with noise as a single agent, develop categories with common features (example, noise level plus temperature level);

(d) Develop cumulative lifetime work history: begin with date of entry into work force, index each job, record date in and date out of each job;

(e) Integrate job exposure to noise and work history for a quantitative estimate of noise exposure "dose" for the individual worker.

Noise Exposure Paradigm II (Ising et al, 1979; Fairman and Johnson, 1979)

(a) Select individuals presumed to be exposed to varying levels of occupational noise on the basis of measurement of the noise level in the worksite;

(b) Have each individual wear a noise dosimeter each workday for an entire week or designated period of time; a microphone of the noise dosimeter should be located in the ear canal and one microphone on the body to measure effective noise doses in the two locations;

(c) Calculate equivalent continuous sound levels for the measurement periods of time;

(d) Supplement occupational noise exposure with off-the-job dosimetry data for 24-hour exposures. Keep work and non-occupational exposures separate. Document noise sources with activity data recorded by the individual wearer.

(e) Develop cumulative estimates of individual exposures through repeated noise dosimetry assessments at defined time intervals supplemented with work history data and recreational and other avocational data.

(5) Identification of selected groups of workers exposed to low noise levels for comparison with high noise and very-high noise exposures inherent in some work environments. A focus on noise exposed groups in industry as opposed to groups exposed to varying levels of community noise may produce the most efficient studies according to the literature.

(6) Systematic delineation and study of factors in occupational environments (in addition to age and sex) which have the potential for confounding noise effects in the study of cardiovascular disease.

4.8 Health Outcome Specification

A major problem in the assessment of the association between noise exposure and cardiovascular disease was the limited number of studies focusing on physiological changes of known clinical significance or relevance to disease development, overt cardiovascular disease manifestations, or changes in known cardiovascular risk factors such as cholesterol (exception was blood pressure). The data most suggestive of a role for noise in the etiology of cardiovascular disease were those demonstrating adverse blood pressure effects. Although the literature suggests that blood pressure changes may occur under continuous exposure to high noise, no studies focused specifically on stroke, one of the major end organ sequella of hypertension.

Individuals known or believed to be at high risk to hypertensive and other cardiovascular diseases were not selected for study as groups in which one might expect to observe adverse cardiovascular response to noise. Examples of such high risk groups are Blacks in the United States who have high hypertension rates, positive responders to exercise stress tests who have been shown to be at high risk to myocardial infarction and offspring of hypertensive parents who are believed to be susceptible to hypertensive diseases, individuals with Type A personalities, individuals presenting with angina that is confirmed by angiographic studies.

4.9 Recommendations Regarding Health Outcomes

Based on our analysis of the extant literature, consideration of the current knowledge of the pathophysiology of cardiovascular diseases and the technologies currently available for diagnosing and defining cardiovascular health, we offer the following recommendations regarding health outcomes in the study of noise effects:

(1) Improvements on the reliability and validity of blood pressure measurements in future studies by standardizing procedures, recording blood pressure measures without knowledge of noise exposure status and by taking repeat readings as opposed to a single casual blood pressure assessment.

(2) Assessment of intra-individual differences in addition to inter-individual differences in blood pressure providing data which are inherently adjusted for many covariables such as diet. It is generally accepted that a shift in blood pressure from low to high even for individuals within the "normal or low blood pressure ranges" increases cardiovascular disease risk.

(3) Reporting of the full distribution of blood pressures as a health outcome to provide a continuous rather than dichotomous variable for analysis with methods sensitive to relatively small differences.

(4) Consideration of hypertensives under medical treatment as a special group of responders.

(5) Special study of stroke in occupational groups since stroke is a major end organ sequella of hypertension.

(6) Focus of future research on a range of cardiovascular responses with measurement of several responses along the disease gradient within any given study. The following disease and physiologic responses may be the most informative based on existing literature (and until appropriate animal models suggest otherwise);

(a) Overt Disease (morbidity and mortality)
    Stroke
    Acute myocardial infarction
    Coronary atherosclerosis as documented by coronary angiography
    Sudden Death
    Hypertension
    Angina pectoris with documentation by angiography or stress testing
    Hyperlipidemia, especially Type II

(b) Physiological Responses (with objective measurement)
    Blood pressure changes
    Lipoprotein changes
    Stress-responses related to the kallikrein-kinin system and prostaglandins
    Changes in platelet function
    Abnormal electrocardiographic response to exercise stress testing

(c) General Measures (subjective measurements)
    General disability
    Symptoms of disease
    Absenteeism from work

(7) Study of special high risk populations including Blacks and offspring of hypertensive parents.

(8) Exploration of indicators which can be used to measure and quantify a gradient of coronary artery disease in populations for investigating the question of the relationship of long-term noise exposure and underlying atherosclerosis. For example, the Rose Cardiovascular Questionnaire may serve to identify symptoms in a large population, catherization and angiographic studies to define degree of occlusion in special groups, and catherization to define coronary disease.

4.10 Sample Selection

Many of the overriding problems which emerged in the analysis of the literature were related to sample selection, sample size and potential selection bias. The potential for such bias is especially high in cross-sectional studies which dominated the literature. Few of the researchers described the population at risk giving details of the sampling methodology, completeness of the sample, exclusions or losses of subjects from the exposure groups and factors selecting individuals into the specific work environments. Nine of the studies which provided reasonably good design and sampling data suffered from other potential sources of bias such as the ecologic fallacy (Knipschild, 1977a; Frerichs, 1980; A. Cohen et al, 1980a; S. Cohen et al, 1981c; S. Cohen et al, 1980b; Manninen and Aro, 1979; Parvizpoor, 1976; Raytheon, 1975 or A. Cohen, 1976; Ising et al, 1979; Meinhart and Renker,1970).

In many occupational studies of the cardiovascular effects of chronic noise exposure there is a strong possibility of selection bias. It is always difficult in an observational study to insure that the two groups to be compared are similar on other risk producing variables while different on the one of interest. If the variables on which individuals differ are measured and there is overlap on these variables between the two groups then various methods of adjustment may be used and a bias-free adjusted comparison between the noise exposed group and non-exposed group can be made. However, if the selection factor is unmeasured or unmeasurable and is associated with both the risk factor and the disease of interest then serious bias can result. In the case of noise exposure it might be that those individuals most sensitive to the effects of noise would refuse to work in high noise areas or would terminate employment due to the noise level. Thus, a comparison of high noise exposed individuals and low noise exposed individuals would be biased to the extent that the high noise group was a select group and those individuals most sensitive to high noise levels were not observed. One way to eliminate this type of bias is to perform an experimental rather than an observational study. If individuals can be assigned to high and low noise categories at random then the only systematic differences between the two groups will be the noise exposure, and thus, any differences in health outcomes can be linked to this exposure. However, in most situations an experimental design is neither feasible nor ethical and other ways of lessening the bias effect must be developed.

Two approaches may be employed to avoid selection bias in observational studies of the health effects of noise. The first procedure, as mentioned above, is to measure other known cardiovascular risk factors and characteristics on which the groups may vary and adjust for them. The possibility of bias due to unmeasured factors remains unknown. The second type of procedure is linked to choice of study group. Attempts could be made to find study groups where individual choice does not determine the noise level later experienced by that individual. Some occupations are such that individuals initially have little choice of the work category with regard to noise exposure and thus the chances of selection bias influencing the results of studies of these workers are minimized. It is important to note that the ability to detect selection bias varies with the study design used. In a classic case-control design, it is essentially impossible to examine or statistically control for selection bias since the "cases" have already experienced selection procedures which are usually unknown. A prospective design allows examination of the ways in which selection could bias the results since the population at risk is defined early. For example, in a prospective study, the rate of transfer from high to low noise areas (or vice-versa) can be determined, the number of cardiovascular events that occur among those who were assigned originally to a high noise exposure area and remained there, those who were assigned originally to high noise and transferred to low noise areas, those who transferred from low noise to high noise, and finally, those who remained in low noise areas over the period of study can be counted. Examination of covariables along with these patterns of change allows examination of many possible biasing effects.

4.11 Study Design and Data Analysis

The summary table of methodological considerations (Table 4-1, p.4-21) indicates that the randomized trial and cohort strategies are the more powerful of the epidemiological designs, and thus are necessary for exploring cause-effect relationships. Data derived from such rigorous designs are not available for judging the strength of the association between noise exposure and cardiovascular disease. Studies by Ising et al (1979), Lees et al (1980b), and Brown et al (1975) are exceptions, and even these researchers failed to apply the more powerful statistical procedures which recently have been developed for the analysis of cardiovascular disease effects other than noise.

Most analyses of the effect of noise on the cardiovascular system have consisted of simple comparison of levels of a cardiovascular disease risk factor such as hypertension between a high noise group and a low noise group. Occasionally the attempt was made to adjust for the effect of possible confounding factors but this was usually confined to age matching or stratification and did not consider or quantify the effects of other possible confounding vairables. The importance of adjusting for covariables in the analysis of the effects of noise on health cannot be overemphasized. A variety of confounding factors must be measured in order to allow investigation of the relationship between these factors, noise exposure levels and the measured health outcome variables. These include the obvious cardiovascular risk factors such as age, race, sex, socio-economic status, and job connected factors such as physical exertion, temperature, humidity, that should be estimated along with noise levels. Table 4-2 enumerates the major cardiovascular risk factors of interest which in any given study may be either confounders or strong risk modifiers. So little data are available in the literature at present that it is difficult to speculate as to which of these factors other than age and sex might be associated with noise exposure, and consequently be potential confounders. In addition, individual factors such as smoking, obesity, behavior type, exercise and hearing threshold levels should be measured. Interaction between noise exposure, health outcomes and hearing threshold levels can be examined. The interrelationships among many variables and noise need to be elucidated. Blood pressure and cholesterol may be important links in the causal network, that is, noise may lead to elevated blood pressure which, in turn, increases the risk of acute myocardial infarction.

Table 4-2

Potential Confounding Variables in Studies of Cardiovascular Response to Noise

Risk Factors	Cardiovascular Response				
	Ischemic Heart Disease				
	Acute Myocardial Infarction	Angina Pectoris	Sudden Death	Stroke	Hypertension
Age	+	+			+
Race/Sex	+WM	+WF		+BF	+BM,BF
Family History +	+	+			+
Socio-economic Status	-				-
Cigarette Smoking	+	+	+	+	-
Sedentary Living	+	+	+		
Exercise	+	+	+		
Obesity	+	+			+
Alcohol - Moderate	-	-	-		
Stress	+	+	+		+
Personality Type A	+				+
Fatigue			+		
Diet					
Sodium					+
Potassium					-
Cholesterol Total	+	+	+		
HDL	+	-			
LDL	+	+			
VLDL	+	+			
Blood Pressure					
Hypertension	+	+	+	+	
Glucose Elevated (Diabetes)	+		+	+	+
Transient Ischemic Attacks				+	
Pre-existing IHD				+	
Biochemicals					
Renin					+
Norepinephrine					+
Kallikreins					-

KEY: Blank = No consistent evidence of association between factor and disease
+ = Factor positively associated with disease
- = Factor negatively associated with disease

In order to investigate the direct effect of noise on blood pressure or on more serious cardiovascular sequella it is necessary to use sophisticated statistical methods developed for other chronic disease epidemiology. These include regression techniques when the outcome is a continuous variable and logistic regression and categorical models when the outcome is not continuous. In addition, methods such as the Mantel-Haenszel Adjusted Chi-square may be used to analyze categorical data without making the assumptions concerning the types of interaction required in other models. Survival analysis based on the Cox model may also be very useful.

As previously indicated, the investigation of any cardiovascular outcome and its relationship to noise should include the collection of information on variables other than noise associated with cardiovascular disease. These variables include age, sex, smoking habits, cholesterol levels, exercise, behavior type and others. If the outcome variable is continuous such as blood pressure, then regression/analysis of covariance models of the following form may be used:

$$\overbrace{\text{(blood pressure) } Y}^{\text{outcome}} = \overbrace{a + b_1x_1 + b_2x_2 + b_3x_3 \ . \ . \ .}^{\text{predictors}}$$

where x_1 - may indicate high or low noise level
x_2 - describes smoking habits
x_3 - age
and so on

With this model we can partition the effects of the various factors and examine the effect of each on blood pressure. A thorough examination of the effects of the associated variables can be accomplished by running subsets of the total set of predictors and comparing the goodness of fit of the model with these subsets. Multiple regression analysis may be used to examine the relationship between hearing loss and health outcomes by using hearing threshold shifts as one of the predictor variables in the regression equation.

Analysis of discrete or dichotomous outcomes such as stroke and myocardial infarction (morbidity and mortality) requires use of categorical models. The logistic model, commonly used in analysis of coronary heart disease data, models the risk of an event as a function of other factors as follows:

$$\mathrm{Ln}\left(\frac{\mathrm{Risk}}{1-\mathrm{Risk}}\right) = B_0 + B_1X_1 + B_2X_2 + B_3X_3 \ldots$$

where the X variables are again predictor variables such as exposure to noise, blood pressure, cholesterol level, etc. An important point to note again, is that the true link between noise and cardiovascular disease may be through an effect of noise on blood pressure. Therefore, care must be taken in the analysis to examine the predictive effects of noise alone and blood pressure alone prior to examining them together, since together neither variable may appear as significant as either one alone.

Life table analysis using the proportionate hazards model as described by Cox (1972) may be useful in the analysis of risk of events for occupational or prospective studies where there is substantial loss to follow-up due to job changes and other associated mobility. In the proportionate hazards model, the relative hazard is used as the measure of association between an event and the possible risk factors. The hazard is the "instantaneous" probability of the event. This hazard must be summed (or integrated) over time to give actual risk. However, comparison among two or more groups can be accomplished using relative hazard analogously to relative risk.

The model structure is as follows:

$\lambda(t, \underset{\sim}{X})$ the hazard depending on time, and $\underset{\sim}{X}$ a vector of covariables

The risk of an event in time T is

$$R(T, \underset{\sim}{X}) = 1-\exp\left(-\int_{o}^{T} \lambda o(t) \cdot \exp(B_1X_1 + B_2X_2 + \ldots B_KX_K)\, dt\right.$$

$$\text{Thus } \lambda(t, \underset{\sim}{X}) = \lambda o(t) \cdot \exp(B_1X_1 + B_2X_2 + \ldots B_KX_K)$$

where $\lambda o(t)$ is the time factor in the hazard model while $\exp(B_1X_1 + \ldots B_KX_K)$ describes the effects of the risk factors. The relative hazard for a unit change in X_i with all the other factors held constant is then $\exp(B_i)$. Thus the antilog of the coefficients in the model can be interpreted as relative hazard similar to that in logistic analysis. Interpretation and use of these coefficients is similar to that of regression or logistic regression. The model usually gives similar results to that of logistic regression, but allows the use of varying follow-up periods and incorporates event-times in the estimation.

Estimation in the relative hazards model is based upon iterative maximum likelihood methods. The time-to-occurrence of an event is included in the estimation, as is that of individuals with incomplete follow-up. This allows use of follow-up data of different lengths of time for different individuals, and thus, may be the method of choice in studies based upon varying occupational exposures.

Another question related to analysis that may be important in occupational studies is how to measure exposure to noise over a long time period. Paffenbarger et al (1975) examined this problem in regard to work activity and coronary mortality. They used person years in the categorization and examined the deaths in each physical activity group relative to the total person years accumulated in that category. A similar model may be useful in the analysis of mortality/morbidity data in groups exposed to different noise levels in the work environment. A common thread throughout these methods of analysis is the need to measure and adjust for possible confounders. This is preferable to matching on confounders (except perhaps age and sex), in fact necessary, since once a factor has been "matched" it cannot be examined in the analysis.

Some of the methods described above such as regression analysis or Mantel-Haenszel adjustment are well known in epidemiological research, but have not been generally applied to investigations of noise and its relationship with cardiovascular disease. Other techniques are recent in development and were developed specifically for the investigations of the relationship between risk variables and chronic disease outcomes. These recent techniques include analysis with the logistic model. Mathematical theory and methods of analysis using this model were developed mainly in the late 1960's and early 1970's in relationship to cardiovascular disease epidemiology and are only now appearing in the main package programs such as SAS. The proportional hazards model, based on the work by Cox in 1972, has only appeared in applied use in the literature since about 1978. Most of the studies of cardiovascular disease and noise were published prior to this time.

4.12 Sample Size Determination

Determination of the necessary sample size for studies of the relationship between cardiovascular disease and noise exposure depends strongly upon the group to be studied and the outcome to be examined. If the outcome is blood pressure measured for both noise exposed individuals and non-noise exposed individuals, then the curve shown in Figure 4-2 shows the power that could be expected for a sample size of 50. These power curves were generated with data from A. Cohen et al (1980a) based on a sample size of 51 in each group, using normality assumptions. They show the probability of detecting a difference in blood pressure between the hearing loss and control groups in Cohen's et al study, but serve as a general example. To have an 80% probability of detecting a difference in systolic blood pressure, the true difference between the hearing loss and control group would have to be on the order of 6.5 mm Hg. Conversely, if the interest is in detecting a 4 mm Hg systolic blood pressure effect, the probability of doing so with these data is only about 45%. For diastolic blood pressure, these data demonstrate that to obtain an 80% probability of detecting a difference, the true difference

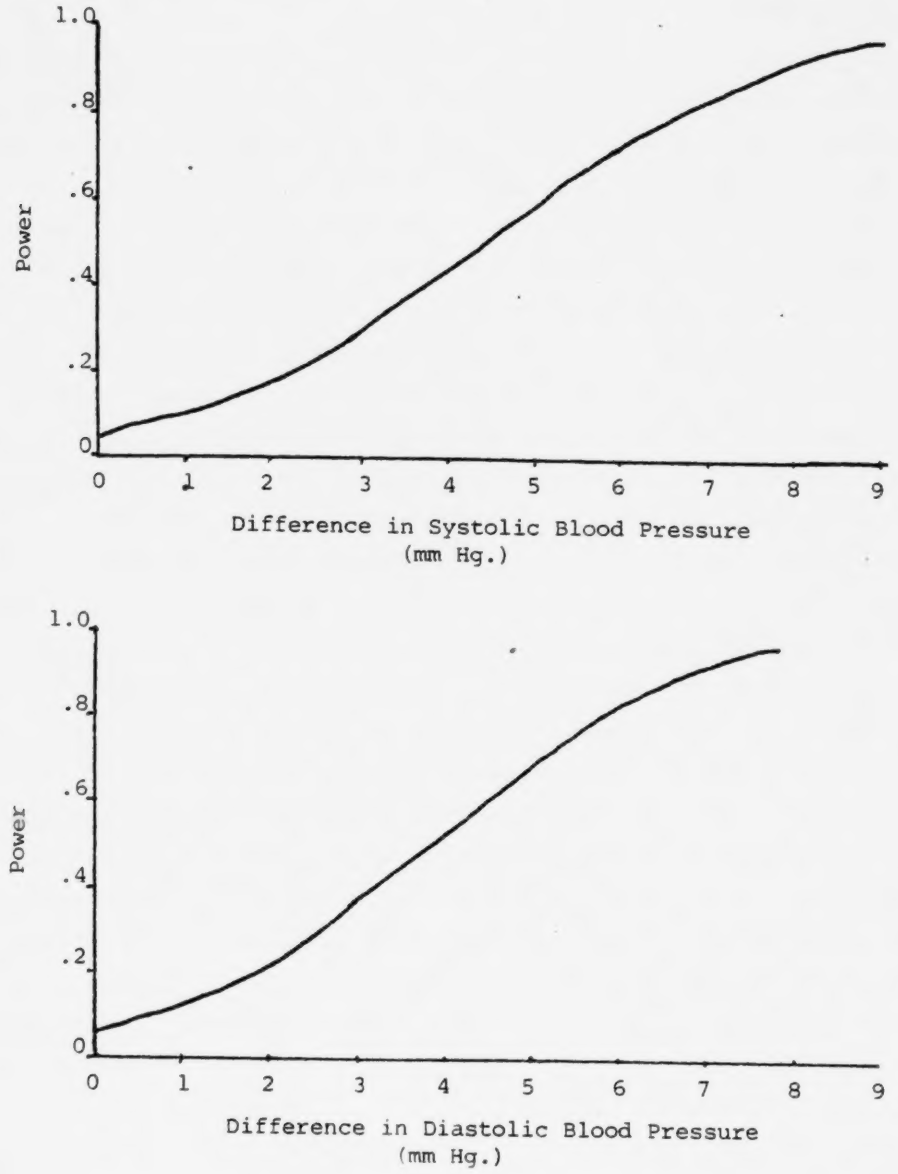


Figure 4-2. Difference in blood pressure required to achieve a given power, under the alternative hypothesis, for a one-sided ($\alpha = .05$) test of the hypothesis of no difference in blood pressure between hearing loss and control group. $n_0=n_1=51$. Data from Cohen, A., Taylor, W. and Tubbs, R.: Occupational Exposure to Noise, Hearing Loss, and Blood Pressure. ASHA Reports 23(10):322-326, 1980.

between the two groups would have to be on the order of 6 mm Hg; conversely, to detect a 4 mm Hg effect in diastolic blood pressure, the probability of doing so is about 55%.

One question that immediately arises is how large a difference in blood pressure is important enough to detect in the noise effects studies. In the Western Collaborative Group Study (1976) the logistic coefficient for coronary heart disease for men was found to be .017. Thus, the approximate relative risk (odds ratio) of coronary heart disease, for men aged 39-48 years, for a unit change in systolic blood pressure was 1.017 [exp(.017)]. The relative risk for a change of 5 units in systolic blood pressure was 1.09 [exp(.017)5]. Similarly, risk of coronary heart disease for a 15 unit change was 1.29. Thus, a change of only 5 units in blood pressure increased risk by 10% in the group. A sample size necessary to detect the shift of 5 units can be calculated, assuming a desired power of .8 and a significance level of .95 using the standard deviation of systolic blood pressure of approximately 13.6 as given in A. Cohen et al (1980a). A sample size of approximately 90-100 subjects in each group would be needed. Since the standard deviation of blood pressure varies for different age and racial groups, sample size would vary depending upon the population subgroups studied.

Examination of discrete cardiovascular outcomes such as stroke or myocardial infarction requires samples of larger size than studies using continuous blood pressure measurements. Assume a case control study nested in a large cohort design where cases are new events of myocardial infarction. Assume an odds ratio for cardiovascular noise effects of 1.5 and a rate of noise exposure in the myocardial infarction group of .5. To obtain a power of .8 at the .95 significance level, 160 subjects in each group, case and control, are needed. However, in order to acquire the 160 cases, an underlying population time factor of approximately 16,000 is needed (16,000 persons observed for one year or 1,600 persons observed for 10 years). This represents the total number of person-years observation necessary to obtain the needed number of cases at a rate of 9.6 coronary heart disease events per 1000 person-years, assuming a cohort of men aged 39-59 years (Roseman et al, 1976).

When noise exposure is less prevalent, the necessary sample sizes will be even greater. For instance, when 20% of the cases are exposed to high noise levels, a sample size of more than 300 cases and 300 controls is required to obtain a power of .8. However, determining noise levels for 300 cases and 300 controls is substantially simplier than determining the noise levels for the complete cohort that could be upwards of 3000 people.

When categorical analysis is required and adjustment for covariables is desired, sample sizes need to be larger than the estimates above. However, if continuous adjustment methods can be used, the total sample sizes remain in this same order of magnitude. Other factors affect sample size determinations. The rates of expected numbers of events used above were based on men aged 39-59. If a study of coronary heart disease events were planned for other age or sex groups, the sample size required in order to achieve an adequate number of coronary heart disease events could be substantially larger.

4.13 Recommendations Regarding Epidemiologic Research Designs

Based on our analysis of current literature the following recommendations are made for population-based research aimed at improving study designs and analysis, thus providing a data base from which causal inferences may be derived:

(1) Careful estimation of desired/required sample sizes need to be made in advance of study implementation for all future investigations.

(2) Priority needs to be given epidemiologic studies employing designs which offer the strongest evidence for associations that may be causal in nature. The designs recommended include:

   (a) Synthetic (or hybrid) retrospective-cohort studies in occupational groups exposed to high noise levels over long periods of employment.

   (b) Large retrospective cohort studies (with continued follow-up) in occupational groups exposed to varying levels of noise.

   (c) Large retrospective cohort studies (with continued follow-up) in selected samples of the population.

   (d) Randomized intervention trials in industrial settings.

(3) Future studies of the effect of noise exposure on the cardiovascular system should adopt quantitative analytical techniques currently employed in cardiovascular epidemiology.

(4) Collaboration of scientists among countries should be encouraged to increase the sharing of ideas and reduce the time that is required for new analytical techniques from one area of investigation such as cardiovascular epidemiology to be applied to noise effects epidemiology.

(5) Any large-scale epidemiologic study should be preceded by an indepth planning phase of approximately one to two years to assure that adequate implementation is feasible.

4.14 Discussion of Recommendations Regarding Epidemiologic Research Designs

4.14.1 Careful Estimation of Desired/Required Sample Sizes Needs to be Made in Advance of Study Implementation for All Future Investigations.

Sample size need not be large, but must be of sufficient size to detect relatively small differences and associated with designs which allow accurate estimates of the antecedent-consequence relationships between noise exposure and disease manifestations/physiologic responses.

4.14.2 Priority Needs to be Given Epidemiologic Studies Employing Designs Which Offer the Strongest Evidence for Associations That May Be Causal in Nature. (Four designs are described.)

4.14.2.1 Synthetic (or Hybrid) Retrospective-Cohort Studies in Occupational Groups Exposed to High Noise Levels Over Long Periods of Employment

Since retrospective-cohort studies may be cost prohibitive due to the large sample size required and the time/effort necessary to reconstruct exposure cohorts, modifications in the design which increase the efficiency without reducing the power substantially can be made. In the synthetic mode, 100% of the cases of the given cardiovascular disease are identified in order to assure an adequate number of outcome events; "controls" are identified by taking a representative random sample of the original cohort of workers. Employment medical records and death certificates provide data for determining the mortality and morbidity (cases); individuals are classified on noise exposures through thorough work histories taken from survivors and from spouses of the deceased. In this mode, estimates of individual noise exposures for a given job can also be derived with use of noise dosimeters by the healthy workers and "cases", if any, who remain in the work force.

The synthetic retrospective-cohort design is cost-effective in that it allows modification and/or extension of the study based on evidence of the effects under observation. If the results of this "case-control" stage are suggestive of an association between noise exposure and cardiovascular outcome, investigation can prodeed to include the total original cohort in the classical retrospective-cohort mode. If data accumulated in this latter analysis are strongly suggestive of adverse effects, a true prospective study can be developed utilizing the remaining healthy cohort of workers and adding appropriate noise exposure and health outcome assessments. (See Scenario #1, Table 4-3.)

4.14.2.2 Large Retrospective Cohort Studies (with Continued Follow-Up) in Occupational Groups Exposed to Varying Levels of Noise

Industrial populations (such as DuPont) with reasonably well-defined past noise exposure information and employment medical examinations would be appropriate groups for study of long-term noise effects given that results generated from synthetic retrospective studies indicated associations worthy of further pursuit.

Table 4-4 outlines the major determinates of an industrial retrospective cohort study. The issues of main importance are population definition, noise measurement, health outcome measurement, confounding variables and the analytical methods to be used to examine the data. Each of these components of a study is contingent upon the others, thus the following discussion, though focusing on each issue separately, will also discuss the interaction between them.

Table 4-3

Scenario #1. Major Components of an Industrial Retrospective-Cohort Study in Synthetic Mode with Stroke as Example

Population	Noise Measurement	Health Outcomes	Confounding Variables	Methods of Analysis
Enumeration of cohort of workers in a specific industry defined by initial date of employment. (20-25 year span). Retrospective determination of all stroke cases and stroke death; random selection of controls (approx. 4 per case) from original cohort file (will include deaths).	Reconstruction of total work histories for job titles and duration of time in job; avocational history of noise exposure from survivors; for deceased, obtain employment and avocational histories from spouse. Specify noise exposure levels of high, medium, low derived from job title (and based on dB and frequency of noise in work area). Ex. High > 85 dB; Medium 70-85 dB; Low < 70 dB	All diagnosed cases of stroke and deaths due to stroke. (Determined from employment files, social security follow-up with verification of completeness against IRS files); Death certificates obtained and cause of death verified by nosologist.	Known factors of age, race, sex Job specific factors ex. physical exertion temperature humidity dust levels chemicals Individual factors ex. blood pressure cigarette smoking history diabetes Transient ischemic attacks Pre-existing IHD	Logistic analysis similar to use in retrospective design with interpretation and inferences in prospective mode since the population at risk is known.

Table 4-4

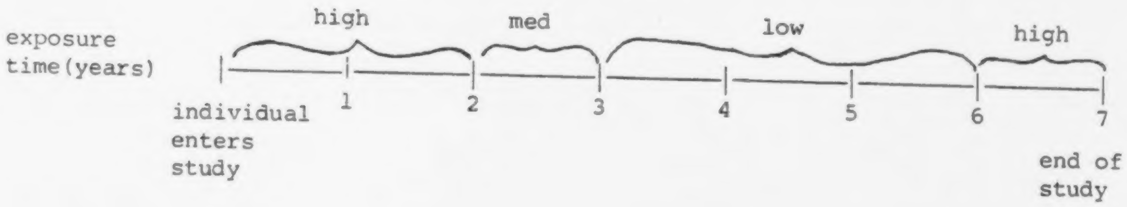
Scenario #2. Major Components of an Industrial Retrospective-Cohort Study

Population	Noise Measurement	Health Outcomes	Confounding Variables	Methods of Analysis
Identified cohort of workers in specific industry defined by initial date of employment (15-20 year span).	Reconstruction of complete work histories for job titles and duration of time on job; specify noise exposure levels of high, medium and low derived from classification of job title (based on dB and frequency composition of noise) Ex. High > 85 dB; Medium 70-85 dB; Low < 70 dB	Mortality from M.I., Sudden Death, and Stroke Morbidity from M.I., Sudden Death, and Stroke on medical records Cholesterol levels Hypertension (under treatment).	Known factors ex. race sex age Job specific factors ex. physical exertion temperature humidity dust levels chemicals Individual factors ex. smoking exercise obesity	Multiple Adjustment methods including - analysis of covariance for continuous outcome variables - multiple logistic analysis for binary outcomes - Chi-square or Mantel-Haenszel Procedures for handling discrete confounders and outcomes - Life-table analysis.
		blood pressure		
		Hearing Threshold Levels		

The population for the industrial retrospective cohort study consists of a group of workers all of whom began work during a known period of time. This population would then be followed through job records, medical records for a substantial period of time. Since we begin with a known and definable population some of the problems of selection that exist in the retrospective or cross-sectional designs would be eliminated. Individual work patterns and vital records would allow the investigator to identify those individuals who do not continue to work in the industry and to examine the covariables associated with attrition. The population is a selected population in that individuals enter the study population through working in a certain industry. Thus one might expect to see a difference on age, race, sex and basic health status between the study group and the general population. This, however, while making generalization to a general population difficult, does not weaken substantially conclusions concerning the health effects of industrial noise upon the workers.

Noise measurement is based essentially on job classification. Each type of work can be examined and classified into low, medium and high exposure categories (preferably by dB and frequency composition of noise). Current and past plant noise surveys can be used to assess noise dose associated with each job. From work histories, a time exposure profile can be constructed for each individual:

Time exposure profile



This type of study still suffers from the difficulty that individuals may change jobs to reflect their reaction to noise, i.e., those most susceptible to noise may by self-selection seek jobs in low noise areas. It is very difficult to determine the extent of this type of confounding which can only be avoided through a randomized clinical trial.

The health outcomes of interest will vary from measurements on cardiovascular risk factors such as cholesterol and blood pressure to disease states of myocardial infarction or sudden death (mortality and morbidity). Measurements of cholesterol and blood pressure levels and other variables of interest may be difficult to obtain if the industry has not maintained a practice of periodic physical exams.

The importance of adjusting for covariables in the analysis of the effects of noise on health cannot be overemphasized. A variety of confounding factors as described previously must be measured in order to allow investigation of the relationship between these factors, noise exposure levels and the measured outcome variables.

In order to analyze the relationship among noise exposure, confounding variables and health outcome, sophisticated statistical methods will be necessary. When the outcome is a continuous variable such as blood pressure, regression analysis, analysis of covariance and analysis of variance will be major tools in the analysis of the effects of noise adjusted for the other variables. Logistic analysis and other log-linear models will be necessary when the outcome is discrete such as death. In addition, methods such as the Mantel-Haenszel Adjusted Chi-square may be used to analyze categorical data without making assumptions concerning interaction.

Sample sizes necessary for this type of study are difficult to estimate without concrete knowledge of the types of comparisons to be examined, the length of follow-up and the base rates of mortality and morbidity in the study group. If periodic physical exams are available, blood pressure adjusted for several covariables can be examined as an outcome with a sample of approximately 200 individuals with different noise exposure patterns. If, however, interest is in more serious and rarer outcomes such as mortality, sample size must be estimated by using the rates of mortality in an equivalent population to estimate the sample size and/or length of follow-up needed. Decisions must also be made as to whether to analyze by person-years of exposure or by individual exposure. This question may lead to use of the proportionate hazards or other models that include "a time at risk" concept more directly in the method. All of the issues discussed above should be examined and explicitly addressed prior to implementation of any study of the effects of noise on the cardiovascular system.

4.14.2.3 Large Retrospective Cohort Studies (with Continued Follow-Up) in Selected Samples of the Population

Populations such as those of the Kaiser Permanente Health Plan and the Framingham Heart Study may serve as excellent general population groups in which to explore the effects of noise on the cardiovascular system. Since reliable blood pressure and cardiovascular disease monitoring currently exists, noise exposure and other environmental exposure assessments may be added to these ongoing studies to increase the cost-effectiveness of noise related research. Careful exploration of the noise exposure data available for reconstruction of the cohorts should precede any major study effort.

4.14.2.4 Randomized Intervention Trials in Industrial Settings

The ideal method for studying the effects of noise on the cardiovascular system would be the randomized trial because of the power of the design in controlling covariables and selection bias. A trial would require a population of workers exposed to high level noise who could be randomly assigned to an intervention group (noise abatement and/or hearing protection program) or a non-intervention group. Such a randomized trial would not be feasible at this point in time because of the known association between noise and hearing loss. Thus, we are forced to approximate this random assignment. This might be accomplished by identifying two areas of a plnat with comparable noise levels and randomizing the areas to a strong noise abatement program or a usual noise protection program. Such a study of long-term exposure to noise would probably suffer from serious problems related to compliance and changes in the work environment and may not be cost-effective until the effects of noise on the cardiovascular system are better delineated. Modified randomized trials offer unique opportunities for studying short-term effects of noise exposure as demonstrated by Ising et al (1979).

4.14.3 Future Studies of the Effect of Noise Exposure on the Cardiovascular System Should Adopt Quantitative Analytical Techniques Currently Employed in Cardiovascular Epidemiology.

To facilitate the adoption of the powerful statistical techniques recently applied to cardiovascular disease data, it is recommended that a position paper on statistical methods for environmental (noise) epidemiology be developed. This paper may be modeled after the work of Breslow and Day (1980) for cancer epidemiology and include detailed examples of applications of the recommended methods. Biostatisticians and epidemiologists currently involved in studies on cardiovascular noise effects and aware of the complexity of the research area should be encouraged to develop the position paper.

4.14.4 Collaboration of Scientists Among Countries Should Be Encouraged to Increase the Sharing of Ideas and Reduce the Time That Is Required for New Analytical Techniques from One Area of Investigation Such as Cardiovascular Epidemiology To Be Applied to Noise Effects Epidemiology.

Much of the evidence suggesting that noise adversely influences blood pressure was derived from industrial studies in Eastern European countries and Russia. Although these investigations appeared to be based on more complex theoretical frameworks than other studies, much detail necessary for replication of the research may have been lost in translation. Collaborative arrangements may be accomplished through linkage with ongoing collaborative cardiovascular studies such as those previously funded by the U.S. Department of State with Poland, East Germany and Russia.

4.14.5 Any Large-Scale Epidemiologic Study Should Be Preceded by an Indepth Planning Phase of Approximately One to Two Years to Assure That Adequate Implementation Is Feasible.

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APPENDIX A

SUMMARIES OF EPIDEMIOLOGIC STUDIES OF THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM

Table A-1
Summaries of Epidemiolcgic Studies of the Effects of Noise on the Cardiovascular System
English Literature
Industrial Noise: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Andriukin, A.A., 1961	Cross-sectional	Industry, Russia	676 males; 556 females a) 307 in tool-making b) 200 in sorting c) 464 in automatic lathes d) 263 in ball bearings.	Unsteady workshop noise a) = 93 dB b) = 103 dB c) = 103 dB d) = 120 dB Exposure: 7-8 hrs. daily in 3 shifts, alternating weekly; 50% workers in same plant for more than 10 years.	BP recorded during work after 10 min. break, on right arm using Riva-Rocci apparatus and Korotkov's method; repeated until constant values; 130/90 taken as upper limit of normal for < 40 years of age and 140/90 for > 40 years of age.
Dega, K., Klajman, S., 1977	Quasi-experimental; pre-post shift measures on exposed & non-exposed	Industry (shipyard propeller grinders), Poland	36 male propeller grinders; 20 workers in similar jobs as controls	Source: pneumatic hammers and high speed grinders; variable frequency; intensity at all stations exceeded N-85 curve. Plotted by dB per octave for 31.5 through 8000 Hz; Duration: 1-14 yrs. for 8 hrs. daily; instrumentation with B&K set.	BP and heart rate; heart minute volume according to Starr's formula; no additional data presented.
Friedlander, B., Grebermann, M., Wathen, G., Zeidler, W. (undated)	Historical-prospective; (incomplete cohort-five year exposure)	Industry, Curtis Bay shipyard, U.S.	441 males, categorized by age and race and by high, medium and low noise exposure; no distributions given Group 1 defined as men having BP reading within one year of initial employment, Group 2: all others.	Shipyard machine shop noise measured by Safety Dept. Noise exposure levels defined as low=<70 dB intermediate=70-79 dB high=>80 dB Duration of exposure: all men had been employed at least 5 years.	Blood pressure defined as: elevated systolic 140 mm Hg and elevated diastolic 90 mm Hg. Data from clinic medical records; no additional information given.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Selection criteria into study, non-response and attrition not stated; specific individual exposures not given; partial control for age; no control for sex, variation in exposure time, medications, comorbidities, etc.	Not stated	No statistical tests given.	"In workshops with intense noise (sorting room, automatic lathes, ball bearings) hypertension is found more frequently than in the less noisy workshop (toolmaking)... In the noisier workshops the morbidity is almost twice as high in persons under 50 years of age...the percentage of patients increase with the duration of employment; particularly marked is the increase after five years of work in the noisiest workshops."
Exposed and non-exposed similar as to mean age and average years employed in shipbuilding. No additional data on control of potential confounders given.	Not stated	Student's t-test	"In workers operating grinders, higher oscillations in the heart minute volume were observed than in workers not exposed to noise." Exposed workers wearing anti-noise ear protectors experienced a decrease in heart minute volume similar to the level in controls.
Incomplete identification and follow-up of a cohort; small and highly selective sample; comparability of low and high noise groups questionable; measurement bias from using extant medical record data. Sample stratified on race and age. No evidence of controlling for any other variables.	Relative risks for race-age groups. For Group 1 RR=2.76 for DBP and 6.36 for SBP - high noise compared to low noise exposure.	Chi-square with Yates correction; Cochran's Chi-square. Relative risks	"The data presented indicates that with prolonged exposure to loud noise of an intermittent nature there is a trend towards both elevated systolic and diastolic pressure when compared to a control population exposed to relatively low-noise intensity: that the relative risk for developing hypertension is greater in those exposed to prolonged loud noise than in controls matched for age and race."

Industrial Noise - Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Gibbons, S.L., Lewis, A.B., Lord, P., 1975	Quasi-experimental; subjects as own controls.	Oil tankers, England	Officers on 4 tankers.	NVR values (dB) ranged from 45 to 96; ships ranked by NRV level.	24 hr urinary 17 ketosteroid levels and urinary volume.
Jonsson, A., Hansson, L., 1977	Cross-sectional	Industry Sweden	196 males; 44 with noise induced hearing loss; 74 with normal hearing; 78 dropped	Surrogate measure: noise induced loss of acuity to 65 dB or more at 3000, 4000, 6000 Hz; assumed repeated and prolonged exposure.	1 measure of BP, supine, after 15 min. rest; hypertension - 160/100. No quality control of observers.
Manninen, O., Aro, S., 1979	Cross-sectional	Industry (Engineering factories) Finland	188 males; 92 females classified by hearing levels	No noise parameter data of engineering production line; surrogate measure, using the frequencies of 3000, 4000 6000 Hz subjects classified: Class I - mild hearing loss, no more than 40 dB at any of 3 frequencies; Class II - moderate loss of hearing 45-60 dB; Class III - severe loss of hearing 60 dB or more. Noise intensity levels and duration of exposure not reported.	Casual BP reading, right arm, in sitting position, at rest; SBP recorded at second sound, DBP at Phase V; all readings by a nurse, apparently "blind" to hearing levels.
Ohrstrom, E., Bjorkman, M., 1978	Cross-sectional	Industry Sweden	75 in machine industry; 99 in 3 textile mills, working in noise 8 hrs. a day; 49 workers exposed 4 hrs. a day	Machine shop: 70-80 dB(A) with peak to background 10 dB(A); Textile mills: 70-80 db(A) with peak background 10 dB(A); 80-90 dB(A) with peak background 5 dB(A); 90-100 dB(A) with peak background 5 dB(A)	Fatigue, vertigo, headache and other symptoms obtained by questionnaire.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
No data other than annoyance as possible confounding variables; volunteer bias; no data on comparability of subjects; small sample	Not stated.	Non-parametric, Wilcoxon matched-pairs test.	Decrease in level of 17-ketosteroids and urinary volume while men were serving at sea compared to mean values obtained when men were on leave. Control values obtained while men on leave were in range of reported values of workers not exposed to noise suggesting officers recover "while on leave."
Groups comparable as to mean age; no controling for age antihypertensive medications, history of disease, etc.	Not stated	1) t-test to compare BP in hearing loss group with normals. 2) X^2 to compare number of hypertensives in the groups.	BP and hypertension was "significantly higher in workers with noise-induced hearing loss" than those with normal hearing.
Mean BP given by sex, and age groups 26-40 and 41-65 years. Relative weight for sex and age group similar in different hearing classes. Excluded those on antihypertensive drugs, with chronic urinary infection or hearing impairment not due to noise damage.	Not stated	Student's t-test for differences in mean BP.	"--Results...indicate that there is a relationship between BP and noise-induced hearing disorder. Prolonged exposure to industrial types of noise first elevates the SBP and to some extent the DBP as well. If the noise exposure, continues, with the resulting severe hearing loss, the SBP tends to return to normal. DBP seems to either rise or fall. Obviously, prospective studies are needed to verify these conclusions."
No data on participation rate. No evidence of controlling for age, sex, duration of exposure although data were collected.	Correlations between noise and symptoms: r_{xy} Headache 0.73 Fatigue 0.96 Annoyance 0.45	Correlations	"Fatigue increases with a higher noise level and a longer period of exposure." Headaches were somewhat less related to noise.

Industrial Noise - Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Parvizpoor, D., 1976	Cross-sectional	Industry Iran	821 male weavers; 412 controls.	96 dBA in weaver group; no documentation of noise levels among controls.	1 BP, sitting after 5-10 min. rest; quiet room; preshift.
Raytheon Report, 1975	Before/after comparison (Hearing Conservation Program)	Industry 4 plants: boiler; nuclear comp., tube mill, superheater U.S.	434 workers in high noise group; 432 workers in low noise group. 44 workers lost to follow-up	Noise in production of large pressure boilers; noise levels given for 75 jobs; Intensity: high noise = ≥ 95 dBA; low noise = ≤ 80 dBA; exposures variable; no ear protection in 1969-70, all workers in high noise area with ear protection in 1972-73. Ear protector usage based on self-report and 1 observation by project director.	Diagnosed medical problems and symptoms from extant records; coders "blind" to exposure conditions; diagnostic criteria not given.
Semczuk, B., Gorns, H., 1971	Mixed cross-sectional/experimental	Acoustic darkroom and worksites. Poland	200 healthy males; (50 in test group; 50 in controls; 100 in worksite)	Intensity 1) audiometer = 100-110 dB for 40 second intervals 2) industry = 80-115 dB "on the average" for 8 hour shifts.	CRIS - index of cardiorespiratory efficiency

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Stratified by age; similar social class; no data on length of employment, sex of controls, previous noise exposure, etc.	Not stated	Tests not given.	Weavers had "significantly greater risk of developing hypertension" than controls; difference appears at "young ages (30-39) and increases with length of employment."
Worker age, job type, length of service, work shift considered in analysis. Previous medical history and use of outside medical services not explored.	Not stated	Wilcoxon matched-pairs, signed rank, test for subjects serving as own control; median test for comparing low noise vs high noise groups.	"Expected relationships between rated usage of ear protectors and incidence of extra-auditory problems were orderly in some cases, but not in others. Methodological shortcomings in determining ear protector usage may have been responsible for this result."
50 controls and 100 test subjects worked in similar temperatures and humidity. No controlling for age, length of exposure, etc.	Not stated	None given.	"In 73% of examined individuals after 8 hours of work in noise of 90-115 dB intensity, the CRIS value decreased while in a control group of 50 workers in less noise, 65 dB, such changes were not observed".

Table A-2
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
Translated Literature

Industrial Noise: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality- Long vs. Short Term	Cardiovascular Response Measure-Quality
Barhad, B., Gradina, C., Mihaila, I., Deculescu, F., Marinescu, V., Cristecu, I., Miclescu, S., 1969	Cross-sectional; pre-post shift	Industry, (Smelter) Rumania	132 workers exposed to noise	Short-term noise effects assessed; levels between 98-127 dB with spectrum extending over several octaves; 100 workers employed for five years.	SBP, DBP, pulse rate; digital plethysmography, coordinator-rhythm strength test; auditory analyzer; new cases of disease; no diagnostic criteria, definitions or measurement details reported.
Britanov, N.G., 1979	Cross-sectional	Industry, (Chemical fiber) Russia	113 women ages 23-49; 3 Groups: I. Spinners II. Warpers III. Twisters	Not specified for each group; 50-100 dB ranges with 63-8000 Hz frequencies. Group I: 90 dB with 125-2000 Hz frequencies Group III: 98 dB workers had 6-10 yrs. of employment.	Blood pressure with hypertension defined as $\geq$ 160/95 mm Hg.; borderline as 140/90-150/95 mm Hg. Complaints No information provided on measurements.
Cieslewicz, J., 1971	Cross-sectional	Industry, (Spinning and weaving) Poland	Weavers: 490 women and 212 men; Spinners: 353 women and 252 men; age groups 18-29, 30-39 40-49, and > 50.	Weavers exposed to noise ranging from N=96-116 for octaves 250-8000 Hz; Spinners exposed at 84-90 dB. Weavers used own ear protectors. Frequency composition of noise for weavers shown; no data for spinners. Most weavers had worked 15 yrs. or more in the mill; no data on spinners.	Hypertension defined as BP > 150/95 mm Hg. Data from periodic examinations of workers. No information on measurement etc.
Demeter, I., Drasoveanu, C., Cherestes, I., Kertesz, I., Demeter, E., 1979	Cross-sectional	Industry (Coal mining) Rumania	100 male coal miners, mean age 45 yrs.; divided into groups by hearing loss. 40 workers had extensive examinations for CV risk profile.	Uncontrolled coal mining noise; 90-105 dB; no additional information given; exposure duration by employment from 1-13 years. Audiometric exam performed, then grouped into normal hearing, incipient sonic trauma and advanced hearing loss.	Sublingual vessel morphology for determining arteriosclerosis; cholesterol levels and blood pressure.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author(s)
No evidence of control for age, sex, weight, co-morbidities, hearing levels of intra-individual variation of BP.	Prevalence ratio of 1.26 for circulatory disease among smelter workers compared to those in motor division.	No statistical tests provided.	"Under the effect of work carried out in noise...most of the workers showed toward the end of the work day a decrease of the systolic and diastolic pressure which varied between 7 and 14 mm Hg. compared to the values found at the beginning of work...The study of the clinical and statistical morbidity of smelting division workers shows, in relation to workers in the motor section, a significantly higher disease rate." (1.26 for circulatory disease) "Our results show that, under the influence of noise and vibrations, changes appear which are shown by disturbances of the functional condition of the entire organism, and not only of the auditory analyzer."
Selection; small sample size; no evidence of controlling for age, obesity or social class in BP analysis.	Not stated; prevalence rate of 2.8 for spinners and twisters compared to warpers	No statistical tests stated.	"In workers exposed to the combined effects of noise at the level of MPL or 5-11 decibels higher and to acetone within the limits of MPC, and also in those exposed to the isolated effect of similar noise, functional alterations of the nervous system following the pattern of neuro-vascular dysfunction and neurotic reactions, and borderline and arterial hypertension, were more frequent. Alterations of physiological functions (visual-motor reaction time, endurance of static work) were more pronounced in workers exposed to the simultaneous effects of noise and acetone than in those exposed mainly to either noise or to acetone."
Selection, selective survival; measurement bias. Data stratified by age; no evidence of controlling for other variables-smoking, shifts, housing, etc.	Not stated; from tabled data, for ages over 50 yrs., prevalence ratio for hypertension for weavers compared to spinners women = 2.2 men = 2.7.	Statistical significance stated; no tests provided.	"The analysis of the frequency of illness among the workers of the weaving and spinning (hard waste) mill has shown clearly that hypertension, neurotic syndromes, and gastric and duodenal ulcers are more frequent among the workers at the weaving department. This difference must be connected with the sharply different working conditions of the two groups and, especially, with the extent of exposure to noise pollution."
Selection bias possible; controlled on age by stratification into < 40 or > 40 yrs. of age; unclear as to how family hx., smoking hx., ponderal index, hypertension were handled in analysis for vessel morphology.	Not stated.	Feinstein's set theory, Venn diagrams.	"The noise plays an inducing role in arteriosclerosis. The arteriosclerosis favors the occurrence of sonic trauma. The occurrence of sonic trauma is earlier in subjects over 40 years of age exposed to noise."

Industrial Noise: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Geller, L.I., Sakaeva, S.A., Musina, S.S., Kogan, IA.D., Belomytseva, L., Ostrovskaia, R., Volokhov, IA.P., Luk'ianova, E., Popova, R.M., Moskatel'nikova, E.V., 1963	Cross-sectional	Industry, (Oil) Russia	1482 workers in oil gases; 366 in oil gases and high noise compared to 263 administrative; 456 physical work.	High frequency 2400-6000 Hz; levels 115-125 dB.	Hypotension defined as < 99 SBP or < 69 DBP; hypertension and cardiovascular neurosis not defined. No other data given.
Gel'tishcheva, E.A., 1980	Cross-sectional; measure during one day and one week and at end of 1 year.	Industry, (Watch assembly) Russia	36 adolescents, 16-18 yrs. old; 11 trainees, 16-17 yrs. old; healthy	Exceeded by 1-4 dB the maximum spectrum (MS) 65, but was not higher than P.S. 70; employed 1-3 years; workers and trainees exposed to similar noise, differed on production stress.	SBP, DBP, pulse rate, EKG, no measurement procedures given.
Graff, V., Bockmuhl, F., Tietze, V., 1968	Cross-sectional	Industry, (Boiler plant) Germany	117 exposed to noise; 50 workers in same plant without noise straining; males without history of hypertension; both groups of same age distribution.	Group of locksmiths, blacksmiths, welders, tube-benders, transportation workers and crane drivers exposed to 90-110 dBA noise of medium to high frequencies. No noise exposure data given for controls of transportation workers without noise strain.	Increase in BP classified into 4 grades according to WHO standards beginning with SBP 150 mm Hg and DBP of 95 mm Hg. Numerous other parameters such as EKG, EEG, clinical examination assessed but not defined. Men with history of hypertension excluded.
Jansen, G., 1959(a)	Cross-sectional	Industry, (Smelting) Germany	669 workers from high noise areas; 336 workers from low noise areas; average age 42 yrs.; response rate high.	Actual noise levels not reported; 34 occupations listed. Workers divided into 4 groups: Groups I & II: Noise < 90dB; Groups III & IV: noise > 90 dB. Subjects had worked at least 3 years under same conditions.	Symptoms of vascular and cardiac problems. By medical exam.: tachycardia, extrasystoles and blood pressure readings. No information on diagnostic criteria or measurement procedures.
Kangelari, S., Abramovich-Poliakov, D., Rudenko, V., 1966	Cross-sectional	Industry, (Motor-testing shop, & foundry) Russia	135 motor mechanics, 152 cleaners; males, aged 20-39 yrs. 30.8% employed ≥ 3 yrs.; 17.3% up to 5 yrs. and 51.9% employed ≥ 5 yrs.; unstated number in control group of fitters.	Motor mechanics = 116-120 dB; fitters = 88-90 dB; cleaners = 100-120 dB.	Sickness rate = cases per 100 based on temporary work disability for 3 yrs. Angina stated as a specific form of illness, but no diagnostic criteria stated. Diseases of nasopharynx, gastritis, ulcers, and myositis also studied.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Selection, selective survival. Stratified by sex and age (age < 40 or > 40).	Not stated.	Not specified.	"In persons exposed to the effect of persistent high noise, arterial hypotension is less common and hypertension much more common than in workers whose work conditions are not connected with the influence of noise. Cardiovascular neurosis is found in a higher number of cases than in employees performing other physical or administrative duties."
Selection bias; bias due to changes in the work environment during the year.	Not stated.	Statistical significance stated; no tests given.	"The intensive delicate visual work performed under exposure to occupational noise not exceeding MS 70 (75 decibels) shows an adverse effect on the functional state of the cardiovascular system in adolescents. The changes of the functional state of the cardiovascular system in the process of occupational activity are more pronounced in the adolescent workers than in the students of VTS."
Selection and selective survival bias; failure to use controls in comparisons for most of analyses; no evidence of control for confounding factors.	Not stated.	No statistical tests described; data presented in graph format.	A larger proportion of hypertensive patients and patients with other heart and blood circulation sickness were found among the noise exposed group than among the workers without noise strain. Within the noise-exposed group, 25-35 year olds, locksmiths and blacksmiths and individuals exposed at least 8 years were most likely to present with hypertension and other circulatory problems. "In the patients with other heart and blood circulation disorders and in the group without pathological findings, low grade, medium and high grade hard-hearing persons were found, but the number of normal hearing persons was lowest in the group of persons with high blood pressure."
Selection bias; measurement bias; groups described on personal, economic, living, working conditions and family life, but no evidence of control in analysis.	Not stated.	Chi-square.	"Vascular disturbances in the extremities, skin findings (paleness) and cardiac findings (rhythm disturbances, tachycardia, extrasystoles) occur statistically significantly more frequently in subjects exposed to high noise levels." "Thus, all this would prove that vascular disturbances, skin findings, and cardiac findings as indicated in workers exposed to noise are caused by noise and thus could be designated as 'noise determined' symptoms or findings. The proof can be based on previous results of experimental noise research."
Selection and measurement bias; no evidence of statistically adjusting for confounding variables.	Not stated.	No inferential statistics; rates (cases per 100) given.	"...that the motor mechanics had a much higher sick rate index than the control group for flu and acute common colds of the upper respiratory tract, as well as diseases of the nasopharynx, breathing organs, and the gastro-intestinal tract." Angina sick rate 8.9 in motor mechanics and 7.8 (cases per 100) in fitters. "...The indexes of the sick rate for the cleaners suffering from vibration illness are higher for every disease than for the cleaners not suffering from vibration illness."

Industrial Noise: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality- Long vs. Short Term	Cardiovascular Response Measure-Quality
Khomulo, P., Rodionova, L., Rusinova, A., 1967	Cross-sectional	Industry, (Type not given) Russia	69 men and 34 women working under high noise; 38 men and 13 women served as controls (low noise) observed over the course of 7 years.	Noise-exposed group: 117 dB, high frequency; Control groups: 60-95 dB low to medium frequency noise. Subjects grouped by yrs. employed as 1-2, 3-4, 5-9, 10-15, 15-30.	Blood serum cholesterol; B-lipoproteins; neurocirculatory asthenia; hypertension; arteriosclerosis. No diagnostic criteria given.
Lanzetta, T., Giovanazzi, A., Furlanello, F., 1979	Cross-sectional (serial EKG measurements)	Industry, (type not given) Italy	12 workers exposed to noise in two work areas; 5 controls, not exposed to noise and of normal hearing. Sex and age of workers not stated. In exposed group, 8 had normal hearing and 4 were hear-impaired.	One work area = 91 dBA; another work area = 101 dBA; 4½ hrs. exposure to usual work noise on the day of testing with 2 successive hours of rest. No other specific noise data given.	Holter (dynamic) monitoring of heart rate; arrhythmias, and ST-T differences (repolarization).
Paranko, Vyschipan, V., Naumenko, B., 1974	Cross-sectional	Industry, (Mining) Russia	103 miners; 61 drillers, 42 sinkers; 2 mines studied; 62 males aged 30-40 years and 41 males aged 41-50 years.	Mine noise of high frequency; workers wore antinoise helmets VTSN-11-T-2M with noise estimates of 85 dB SPL based on previous measurements.	Arterial blood pressure with increase defined as: 130/90 mm Hg at ages < 40 yrs.; 140/90 mm Hg at ages > 40 yrs.; heart rate quickening defined as > 80/min., slackening to < 70/min.
Pilawska, H., Mikulski, T., Rosin, J., Soroko, M., Wysocki, K., 1977	Cross-sectional	Industrial, (Large shipyard) Poland	1826 exposed to noise; 5825 working in low noise area; 67.5% of employees included; men and women.	Multiple source shipyard noise of > 85 dB L_{QM} for longer than 5 hrs. a day for exposed workers and < 75 dB L_{QM} for non-exposed workers. NPL method of measurement used; plant divided into 3 parts with each part measured separately using 9 daytime and 12 nighttime points.	Hypertension; no diagnostic criteria, no definitions; from periodic health exam. Records of 13 doctors in the industrial clinic. Also studied were hearing damage, stomach ulcers and psychic problems.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Selection bias; age, length of employment, specialization, and state of the nervous system were controlled in analysis. Nutrition controlled on day of blood testing.	Not stated.	p-values, no tests stated.	"Industrial noise with an intensity of 95-117 dB acting during the course of 5 years or longer leads to impairment of the metabolism of lipids in workers. The disorders were manifested as hypercholesterolemia and a tendency toward an increased quantity of total lipids and beta-lipoproteins in blood. The degree of increase in the cholesterol content in the blood depends on the length of employment in conditions of intense industrial noise. After 5-15 years of exposure to industrial noise, endogenous hypercholesterolemia, more pronounced in persons with functional disorders of the nervous system, developed in the majority of the workers."
Multiple environmental and personal variables such as smoking monitored. Unclear as to how these were used in analysis; selection bias potential.	Not stated.	Serial data presented in graphic and tabled format. p-values stated; no specific methods given.	"The increase in the cardiac rate was indeed constant in the groups 'exposed' with normal auditory capacity, was maintained throughout the work shift, and re-entered the normal limits only after cessation of work. The results were statistically significant in comparison with the group of workers 'not exposed' to noise and with those with hearing impairments. It is interesting to observe the persistence of the response in the increase in cardiac rate (under exposure) to environmental noise, even after many years of exposure to this noise."
No controls or control group used; no statistical control for potential confounders such as age; selection bias, selective survival and selective recall.	Not appropriate.	Not stated.	"Thus, the results presented show that in miners exposed to the effect of vibration and noise at levels allowable by hygienic norms, stable functional shifts develop. However, their development occurs more slowly than in those exposed to the effect of vibration and noise of high parameters."
Potential non-respondent and selection bias; no evidence of controlling for age, sex, treatment, co-morbidities or length of employment with hypertension data.	Prevalence ratio for hypertension among exposed compared to non-exposed = 2, (not controlled for age).	Frequencies compared; no statistical tests given.	"Significantly higher numbers of differences in the state of health detected in workers during periodic examinations, such as hearing damage, ulceration of the stomach and duodenum, and psychic disturbances and neurosis in the group of workers who were in the zone of high noise level during work, were confirmed."..."Smaller differences, but also statistically significant occur in the frequency of hypertension found in both groups (more than double in group A)."

Industrial Noise: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Rumiantsev, G.T., Mekhelson, D.A., Sechenov, I.M., 1971	Cross-sectional	Industry, (Ships) Russia	86 sailors; 25-35 yrs. of age with > 3 yrs. employment, 8 ships	Steady noise of high speed engines. Group 1 - (engine squad): low and medium frequencies; Group 2 - low and medium frequencies and diet supplement; Group 3 - crewmen (controls).	Blood cholesterol, blood sugar, B-lipoprotein level: λ - globulin level; blood pressure and pulse.
Sanova, A.G., 1975	Cross-sectional	Industry, (Compressor shops) Russia	144 male compressor operators, 20-50 yrs. of age; 30 controls from the same plant working under low noise.	Actual noise levels not reported. In compressor shops 87-98 dBA and 90-110 dB infrasound levels. Duration of exposure in years employed ranged from ≤ 1 to ≥ 20 years.	Arterial blood pressure, EKG, contractile and minute blood volume, peripheral resistence. Definitions and measurement procedures not stated.
Shatalov, N.N. 1965(b)	Cross-sectional	Industry (Type not stated) Russia	1019 exposed to continuous noise and 338 to intermittent noise; also for special studies: Group 1: 368 in continuous noise; Group 2: 221 in intermittent noise.	No information provided on noise exposures for group of 1357 studied for BP; For Group 1: 85-122 dB, wide band spectrum with high frequency domination; continuous noise. For Group 2: 85-111 dB, wide band spectrum with high frequency domination, intermittent noise	Blood pressure taken at beginning of work day after 10 minute rest. High blood pressure defined as > 130/90 mm Hg for persons < 40 yrs. old and > 140/90 mm Hg for those > 40 yrs. old. EKG, ballistocardiography, phonocardiography, peripheral resistence, venous pressure, heart size. No definitions or criteria given.
Stasiow, A., Guzikowska, H., Guzikowski, J., 1974	Cross-sectional data; pre-post shift measures	Industry, (Mine) Poland	31 workers in the washers, a mechanical coal processing section of a mine, ages 35-62 years; employed > 6 years. No controls.	Noise levels ranged from 86-102 dB; 7.5 hours exposure daily; noise measurements in the work place, in a position near the head of the worker.	Arterial BP, in sitting position, with mercury sphygmomanometer, after a night's rest, before work and repeated after 7.5 hrs. of noise exposure; cold pressor test; EKG at rest with 12 standard leads and evaluated by Minnesota Code.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Self-selection; bias due to non-comparable comparison groups; no evidence of controlling for age, diet, exercise, diabetes, weight, etc.; non-response or missing data bias.	Not stated.	p-values given; no statistical tests stated.	"Thus, significant increase of the sugar concentration in the blood on the 15th day of the course and of B-lipoproteins in the blood serum on the 30th day of the course in sailors of the 1st group, with no analogous shifts (during the same period) in persons of the 3rd group, can probably be treated as the consequence of noise-vibration effect. The changes of the arterial pressure and pulse before and after the watch in the sailors of the 1st group can be explained in the same manner."
Small sample size bias; selection and measurement bias; groups stratified by age; no other confounding variables considered.	Not stated.	None stated; very little data presented.	"The analysis of the indexes of the functional state of the cardiovascular system indicated that the percentage of workers with normal blood pressure decreases the longer the length of employment in compressor shops." ..."In the basic group (noise), we observed the increase of systolic pressure to be more pronounced in relation to age (than in workers not exposed to noise)."..."The reduction of the contractile function of the myocardium, increasing with the length of employment in the compressor shop, can be related to the disorder of the functional state of the myocardium under exposure to noise."
Selection and measurement bias. No evidence of control of any confounders such as age and sex. Individuals with history of heart disease excluded from study.	Not stated.	No statistical tests stated.	"Very often the workers in 'noisy' occupations have complaints of a cardiac character; their arterial pressure is changed. In persons exposed to continuous noise, first of all the symptoms of vascular dysfunction are observed (lability of the arterial pressure, tendency toward the reduction of venous pressure and the reduction of peripheral resistance, bradycardia). Under exposure to intermittent noise in the course of a work day a clear tendency to hypertension is present (rising arterial blood pressure, increased rapidity of dispersion of the pulse wave in the elastic vessel, the symptoms of capillary spasm more common)."
Self-selection; interaction of selection and exposure to noise; regression to the mean.	Not stated.	Test for signs.	"In the examined group of workers, under the influence of exposure to industrial noise, a significant increase in the diastolic pressure and a spastic state of the arterial vessels on the periphery were confirmed. It was shown that exposure to industrial noise causes an increased vessel reactivity. The observed changes in the EKG curve can speak for the domination of the parasympathetic system."

Industrial Noise: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality- Long vs. Short Term	Cardiovascular Response Measure-Quality
Tavtin, IU. K., 1976	Cross-sectional	Industry, (Machine building plant) Russia	861 workers divided into 5 noise groups; 281 women to 580 men; employed < 5 yrs. to > 20 yrs.	5 noise exposure groups. Group 1: 121 workers, in 70 dB noise; Group 2: 139 workers, in 83 dB noise; Group 3: 168 workers in 94 dB noise; Group 4: 267 workers in 110 dB noise; Group 5: 166 workers in 114 dB noise.	Clinical examinations with diagnoses of functional disorders of the cardiovascular and nervous system, hypertension and arteriosclerotic heart disease.
Terentiev, B.G., Sheludiakov, E.E., Sviridora, E.S., 1969	Cross-sectional with no controls; before-after work measures; anecdotal; experimental.	Industry, and laboratory (aviation) Russia	90 engineering and technical staff; 15 healthy men.	Separate conditions of 100-102, 110-112, 118-120 and 130-136 dB; subjects exposed either daily or 2-3 times a week for 1-6 hrs. each time. Laboratory - exposure at 1 hr., 3 hrs. and 6 hrs.	Blood pressure; pulse rate; EKG changes. No diagnostic criteria or definitions given.
Volpilkina, G.I., 1959	Cross-sectional; before-after work shifts.	Industry, (Textile) Russia	300 spinners, 300 weavers No. of unspecified controls.	No measurements provided; subjects were spinners, weavers and controls were workers in packing and mechanical plant.	Blood pressure. No information provided on measurement.
Zvereva, G.S., Onopko, B.N., Ratner, M.V., 1975(a)	Cross-sectional; (before-after work shifts). No controls.	Industry, (Dolomite-Flux plants) Russia	334 workers in 8 professional groups, aged 30-40 years.	Quarries, excavation and drilling places: 95-100 dB, low and medium frequencies. Crushing-enriching plant; 86-106 dB, predominate high frequency components. Noise measured in 164 work places.	Arterial pressure; state of the capillaries; complaints such as heart pain and fatigue; asymmetry of arterial pressure, anisocoria and trembling of the hands.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Selection and selective survival; impure noise exposure groups; no evidence of control for any variables including age and sex.	Not stated.	No data presented; no statistical tests.	"Under exposure to continuous noise of 110 dB...significant increase of the functional disorders of the nervous (12.5%) and cardiovascular (6.3%) systems was observed. In the exposure to a pulsed noise level of 114 dB,...functional disorders of the nervous system in 16.2%, functional disorders of the cardiovascular system in 8.4% of the cases." "...A significant increase of functional disorders of the nervous and cardiovascular systems take place under exposure to noise of a general level of 110 dB when additional neuro-emotional factors exist, and under exposure to pulsed noise of 114 dB."
Selection; impure exposure group; no control group; no evidence of analysis of data in a before-after mode.	Not stated.	None given.	"...we are able to conclude that a one-hour per day exposure to noise in the 110-112 dB range can be successfully endured, but that daily exposures of 3 and 6 hours will bring about harmful effects. Research concerning reactions to noise in the range of 118-120 dB showed that the character of the changes following a single exposure is the same as described above. However, the alterations are more pronounced and the after effects persist longer. Especially severe reactions were observed during exposures lasting 3 and 6 hours." "...The frequency of heart contractions decreased by 16-17 beats a minute, maximal and minimal arterial pressure diminished, as a rule; the beat and minute volume of the heart, which were only slightly affected by exposure to noise of lesser intensity, decreased markedly upon impact of noise of the 118-120 dB level; the reaction of the cardiovascular system to physical exertion increased." ...During daily exposure of 1, 3, 6 hours of noise at 120 dB level, an increase in systolic and diastolic blood pressures were observed.
Selection; non-response; measurement bias. No evidence of control of any variables other than stratification by age.	Not stated.	Not stated.	"It is clear from our documentation that an unfavorable microclimate and work-related stress do provoke characteristic disturbances in the reactions of the cardiovascular system and the higher nervous activity among the women working in the textile industry. This should give occasion to the elaboration of new prophylactic measures which would aim to improve further the working conditions of women."
Selection bias; bias due to lack of controls and non-comparable subjects within the study groups.	Not stated.	Not described; very little data provided.	"The data obtained show that noise and vibration in the plants of the flux-dolomite industry have quite high levels and cause changes in hearing and disorders in the function of nervous and vascular systems typical of vibration-noise pathology."

Table A-3
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
English Literature

Industrial Noise: Some Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Antonova, K.P., 1979	Cross-sectional Pre-during-Post-shift measures	Industry Russia	33 male workers; no controls.	Ore-dressing plant noise 92-112 dB mills; 97-104 dB separators; no details provided; employment 3 months to 11 years. Vibration measurement presented in detail.	Pulse rate, brachial and temporal arterial pressure. No data or measurement procedure provided.

Cuesdean, L., 1977	Cross-sectional	Chemical industry	160 exposed to noise
			160 not exposed (149 males & 11 females in each group)
				Intensity: air compressor operators = 100-106 db;
				stokers = 100-106 db;
				mechanics = 95-100 db;
				electricians = 90-95 db;
				lab assistants = 85-95 db.
				Exposure duration - 6 years in 21-40 year old group; 10 years in 41-60 year old group. Measurement 2 hrs. after an 8 hr. morning shift; in winter.
					ECG on 12 leads; Alterations classified by criteria of the Minnesota Code.
					BP measured during first hr. of work and at end of shift. Hypertension defined as > 140/90 mm Hg.
					Hearing loss.

Hannankari, I., Jarvinen, E., Partanen, T., 1978	Cross-sectional and historical/prospective with subgroups	Industry (Railway workers) Finland	437 locomotive engineers exposed to railroad noise; 1575 trainmen, 1224 railroad clerks as reference groups.	Intensity: 45% of the measured noise levels exceeded 85 dB (A) during 0.5-2 hr. measuring period. Exposure from Dec. 1955 to Dec. 1973. In follow-up analysis all engineers, every 2nd trainmen and all railroad clerks employed on Dec. 1, 1955 were included.	Reported on questionnaire any diagnosed hypertension, M.I.; mortality data from circulatory diseases.
Kavoussi, N., 1973	Cross-sectional	Industry Silo Iran	465 males	Not measured, but "workers could not ever understand each other when speaking." Employment 10 years, 11-25 years over 25 years.	Average of 3 BP readings using a portable Vaquez apparatus. BP taken in AM before work, after being seated 1 hr. and resting supine for 5 mins. Hypertension defined as 140/90 or greater. No information about data collectors or reliability of measurements.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported by Author
Individuals measured at several points in time - no mention of controls	Not stated	None given	Brachial and temporal arterial pressure rose in mill operators, mill operator assistants had 2-6mm increase in brachial arterial pressure whereas separator operators showed no significant change in arterial pressure.
No data provided as to total noise exposure, how subjects were selected or completeness of sample. Relative humidity of exposed groups varied, no data given for controls. Groups compared on smoking, overweight and diet excess in animal fat but no analysis. Sex, age, SES and physical activity not controlled.	Not stated	Not specified	"...Data indicates an increased frequency of ECG alterations at rest. ...Indices 3-1, 3-3, 9-2 were more frequent among the men working in a medium of permanently intense noise. The highest incidence was that of upward deflection of segment ST, of the benign type. The alterations detected might be assumed to evolve towards organic lesions. Intense permanent industrial noise causes...a high incidence of cardiovascular ECG alterations that can be statistically interpreted."
No data on controlling provided.	Not stated	Observed and expected deaths calculated, Chi-square test.	Cross-sectional data showed no significant findings on CVD. "The evidence on the relatively high risk of disease of the circulatory system and tumors of the engineers during the follow-up period was, as summarized, well established...According to the results of the study, technical improvements which lower noise and vibration (are recommended)."
No controls, no data as to previous or concurrent noise exposure, health conditions, hearing thresholds, ear disease, etc. 235 or 34% of workers not included because they "were in administrative positions or absent." Workers stratified by age (≤ 39, 40-54, 55-64). Labor turnover during the 34 year operation of the plant was "very low."	Not stated	% of hypertension by age and length of employment. No statistical tests.	1) ...There is no case of abnormally high BP in men up to age 39. "This means that the length of exposure to a noisy environment does not induce high BP in men under 40 years of age." 2) "After 40 years of age when there is a greater tendency to develop high BP...the length of exposure to a noisy environment can be an additional factor in inducing this condition." 3) In older age group "there is greater incidence of high BP because age and length of exposure are greater than in the previous categories".

Industrial Noise: Some Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Mosskov, J.I., Ettema, J.H., 1977b (III)	Lab experiment with industrial noise	Controlled laboratory The Netherlands	12 healthy males; 19-26 yrs. old; served as own controls.	Intensity: L_{eq} 98 dB(A) Taped noise through headphones in soundproof room; sessions in random sequence of adaptation, rest, exposure, rest. Total duration of noise 30-40 sec.; highest intensity for 6-10 sec. Duration: Three 15 min. periods exposure with 2 rest periods of 5 min.	BP by cuff method, heart rate, pulse pressure, respiratory rate, sinusarrhymia.
Proniewska, W., et al, 1972	Cross-sectional	Industry Poland	121 females in noise areas; 9 controls in quiet areas.	Intensity: 110 dB, frequency ranged from 31.5 Hz to 16,000 Hz; temperature 24°C; relative humidity 78%; work experience from 1-10 years.	Total serum lipids; beta lipoproteins, total cholesterol, FFA.
Yazburskis, B.I., 1971	Quasi-experimental; ultrasonic lab under work conditions	Industry Russia	36 healthy workers; 21 males, 15 females; a) 10 at ultrasonic disperser of 20 kc; b) 14 at 18 kc emitter; c) 12 at 8 kc emitter	Source - RUZ ultrasonic disperser of 20 kc, emitter of 18 kc. Intensity: a) up to 160 db; b) up to 100 db; c) up to 132 db. Exposure in work 4-5 hrs./day. Instrumentation and measurement not given. Subjects working in ultrasound 2-5 yrs.	ECG and BP; no details given.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Random assignment of noise type used; no consideration of BP lability in small sample.	Not Stated	Wilcoxon Test	"Increase of DBP and of respiratory rate and decrease pulse pressure and heart rate observed."
Subjects on optimal diet; ages ranged from 20-40 years; cross-sectional data treated as serial measurements.	Not stated	Student's t-test and student's paired test for serial data of 9 controls.	In first 2 hour interval the cholesterol, beta-lipoprotein and FFA were raised with a decrease in total lipids. In second 6 hour interval, only an FFA increase was observed.
No evidence of randomization or statistical control of variables.	Not stated	None stated	Workers engaged in the operation of high-power acoustic units displayed reduced heart rate, enlarged T-wave, diminished P and R waves and reduced systole: diastole ratio. Exercise caused a downward shift of the RS-T segment of the ischemic type immediately after work with ultrasound. BP fell towards the end of the workday and did not return to its initial level 5 minutes after an exercise tolerance test in all groups.

Table A-4

Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System

Translated Literature

Industrial Noise: Some Adverse Effects Reported by the Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Andrukovich, A.I., 1965	Cross-sectional	Industry, (Bendery silk combine and textile weaving factory) Russia	846 female spinners & weavers, 16-49 yrs. of age, 1-5 yrs. employment compared to 8972 women in the population of the geographic area.	High frequency noise with intensity of 99-102 dB in weaving and 87-88 in spinning.	BP measured 4-6 x by Korotkov-IANovskii method, with Riva-Roçci mercury manometer, right arm, 15-20 minutes after 1st shift. Taken over yrs. 195[illegible]-1963. Hypertension defined as 140/85 mm Hg; Hypotension ≤ 90/50 for 16-19 yr. olds and ≤ 100/60 mm Hg for 20-49 yrs.
Capellini, A., Maroni, M., 1974	Cross-sectional	Industry, (Chemical) Italy	1286 men, 60 women, 98.6% of employees.	No information provided. Implied periodic instrument samplings.	Hypertension: SBP > 160 mm Hg and DBP > 90 mm Hg. Coronary disease: Myocardial infarction diagnosed by specialist on history and/or EKG and VCG; angina pectoris: abnormal S-T response to exercise.
Folprechtova-Stenzlova, A., Janicek, M., 1966	Cross-sectional	Industry, (Foundry) Czechoslovakia	944 male workers classified by noise area.	Foundry Noise: Group A: 74.8 to 95.1 dB; average = 92.3 dB (Lin); Group B: 103-124 dB; average = 107.9 dB (Lin) Subjects are classified by exposure: < 1 yr.; 1-5 yrs.; 5-10 yrs.; > 10 yrs. employed. Noise (acoustic stress) measured with SLM, type B & K 2203 and B & K analyzer type 1613.	Blood pressure; lowest value of 3 readings used; taken in sitting position, on right arm, toward end of workshift, by Korotkov method; one observer.
Grusha, A.N., 1974	Cross-sectional, patient series.	Industry, (unknown, neurological unit, Chernikhov Regional Hospital) Russia	134 noise-exposed patients with neurosis like states; 100 patients with closed craniocerebral injuries; 110 patients with infectious arachnoencephalitis.	134 patients with neurosis like states "as a result of a long-term effect of high frequency industrial noise". No further details given.	Brachial arterial pressure measured by Korotkov-IANovskii method; temporal pressure by Markelov method; central retinal arterial pressure (CRA) by Baiiar method. No details given.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
No data as to selection of control group; possible selection bias and bias due to attrition over the 5 yrs.; stratified into 5 yr. age intervals.	Not stated.	Frequencies compared; statistical tests not shown.	"Arterial pressure was high in female weavers compared to the mean age-specific indexes for arterial pressure in women of the control."..."The processing of the data obtained demonstrated the statistical significance of the higher levels of the systolic pressure within the age groups 16-19, 30-39 and 40-49 years."
Age controlled by stratification. Blood pressure and sex not controlled in myocardial infarction data.	Regression coefficient not provided.	Chi-square; weighted regression after logistical transformation of disease data.	The data indicate that the risk quota for coronary disease due to exposure to intense noise is equal to that in the non-exposed population by an increase in age of 10 years. No significant correlations were observed for hypertension.
Potential bias of non-response, selective survival, selective recall. Multiple variables available for analysis: age < and > 40 yrs., no. of yrs. employed, shift, smoking, no., of children, time commuting to work, weight, sleep, salt intake, diet (fat, veg., fruits), amt. of fluids consumed daily. Apparently only age was taken into account when single indicators were evaluated.	Not stated.	Student t-test. No evidence of multivariate analysis.	"Different levels of acoustic stress do not affect the level of BP if exposure (duration) and schedule are kept constant. If different exposure lengths and schedules are introduced, workers with a longer exposure do have a higher BP, and workers with a 2 or more shift schedule do have a lower average BP." (Finding not consistent across ages.) "Our results seem to suggest that it may be next to impossible to evaluate the particular factors in isolation, and that results should always be seen as the outcome of the working and living environment as a whole. The difference between the two age groups can be explained by the effects of age itself or by the different time factors, combined with other particular causes."
Apparently no control group for comparison; no evidence of controlling for unequal sex distributions of groups; no age data provided; potential selection bias.	Not stated.	None stated.	Of patients (134) with neurosis-like states from long-term effects of industrial noise, a greater proportion suffered from temporal arterial hypertension and central retinal arterial hypertension than from brachial arterial hypertension.

Industrial Noise: Some Adverse Effects Reported by the Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Ising, H., Gunther, T., Havestadt, C., Krause, Ch., Markert, B., Melchert, H.U., Schoknecht, G., Theseld, W., Tietz, K.W., 1979	Cross-sectional and intervention (with and without hearing protectors).	Industry, (Brewery) Germany	90 volunteers; 36 working in noisy areas & 54 in areas of less noise; controls studied for 1 day; 18 noise-exposed workers examined for 2 days and 12 workers examined for 2 weeks while wearing hearing protectors half of the time.	Noise levels given for each job in 6 work environments. All workers wore individual dosimeters: bottling cellar workers = $\bar{X}$ of 95 dB(A) ± 0.7 dB; control group = $\bar{X}$ of 82 dB(A) ± 1.2 dB.	Blood pressure taken with semi-automatic device; subject sitting, at end of shift, $\bar{X}$ of 4 BP readings taken as measured value. Blood and urine tests: epinephrine, norepinephrine, cholesterol, magnesium, etc.
Jansen, G., 1961(b)	Cross-sectional	Industry, (Smelting) Germany	669 workers from high noise areas; 339 workers from low noise areas; average age of 42 yrs.; no chronic diseases.	High noise group exposed to more than 90 dB(B); low noise group exposed in noise not less than 65 dB(B); average length of employment 11 years.	Symptoms of vascular and cardiac problems; tachycardia; extrasystoles.
Jirkova, H., Kremarova, B., 1965	Cross-sectional	Industry, (Large-engineering factories) Czechoslovakia	34 noisy workplaces = 766 men and 203 women. 6 not-noisy workplaces = 371 men and 318 women.	Noisy areas ranged from 85-115 dB for greater part of the day with noise considered disturbing; not-noisy areas had dB levels of < 70 and noise not disturbing.	Medical record data indicating hypertension, subjective complaints, absences from work due to illness. No diagnostic criteria or definitions reported. Data collected by plant physicians with no standard procedures.
Kachnyi, G.G., 1977	Cross-sectional	Industry, (Cloth combine plant) Russia	591 female weavers from 15-27 years of age divided into 2 groups by noise levels and 20 healthy controls working in adm.	High frequency noise from looms. Group 1: 106-108 dB; Group 2: 102 dB; duration of employment Group 1: 17 for 3-6 mos., 172 for 1-5 yrs., 111 for 6-10 yrs.; Group 2: 18 for 3-6 mos., 164 for 1-5 yrs.; 109 for 6-10 yrs.	Arterial and temporal BP. Arterial taken with Mercury manometer , on both arms, middle of 1st shift of last of a 5 day work week; hypertension $\geq$ 140/90 mm Hg; hypotension < 100/55 mm Hg. No information regarding quality control of measurement.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Selection; possible Type II error due to small sample size; no statistical controlling on age, sex, weight, smoking.	Not stated.	t-test.	"When working without ear defenders at a mean exposure to noise of 95 dB(A), the systolic blood pressure was higher by almost 7 mm Hg ($\alpha < 0.001$) and the excretion of vanillyl mandelic acid in urine was higher by 67% ($\alpha = 0.013$) and that of noradrenaline by 16% ($\alpha = 0.05$) than when working with ear defenders. The actual daily average noise level reduction of this device was 13 dB. After one week of work without ear defenders magnesium concentration in the blood of 12 test persons was by 5% ($\alpha = 0.05$) lower than after one week of work with ear defenders. The evaluation of the parameters of 26 test persons showed a negative correlation of $c = -0.52$ ($\alpha = 0.003$) between the magnesium content of blood sediment and the increase in blood pressure when exposed to noise." ..."The comparison of noise workers and a control worker group, however indicated no (statistically) significant difference." ..."Only the examination of the same test subject under two or more different noise stress conditions is suitable for proving the effects of noise with statistical significance."
Selection and measurement bias; no variables controlled in analysis.	Not stated.	Results reported as statistically significant; no tests given.	"The total result of the examination showed that no defined noise disease could be determined, with the exception of hearing damage."..."The vegetative disturbance is stronger than had been suspected; it occurs especially with wideband noises and can be detected in persons at rest and engaged in physical activity. A comparative examination of 1005 persons employed in the smelting industry confirmed these findings."
Selection bias probable; stratified by sex. Age partially controlled. No other confounders considered in the analysis.	Not stated.	Chi-Square.	"The results of the comparison show a higher occurrence of hearing damage, subjective complaints, hypertension, and peptic ulceration in persons exposed to noise." On the other hand, the authors did not show that the frequency of the diseases investigated, with the exception of hearing damage, depended on the length of employment. Findings were not consistent for men and women.
Controlled for other industrial noise exposures by selecting women with 1-2 yrs. work on collective farms or in quiet offices.	Not stated.	No inferential statistics employed.	"Hypotension was more frequently observed with a shorter length of employment." 64.7% hypotensives were noted among weavers in Group 1 and 50% among Group 2 with 3-6 months employment. "Our data confirm the opinion of M.M. Pokrovskii (1968) that a lowering of the arterial pressure occurs frequently in young persons." The number of hypertensives increased with the length of employment. Deviations of the value of the temporal-brachial coefficient from the norm were not observed.

Industrial Noise: Some Adverse Effects Reported by the Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Kalicinski, A., Straczkowski, W., Nowak, W., Proniewska, W., Ro'zan'ska, T., 1975	Cross-sectional	Industry, (Spinning and weaving) Poland	140 women 45-51 years of age; $\bar{X}$ age = 49; compared by years of work in noise: Group I = 31 workers; Group II = 48; Group III = 61 workers.	Steady noise from spinning and weaving industry at frequencies between 32 and 16,000 Hz with intensities 95-105 dB; women grouped by time worked in noise: Group I = 1-6 years, Group II = 7-12 years, Group III = 13+ years worked.	BP taken after 30 min. rest; hypertension defined as SBP > 140 mm Hg and DBP > 90 mm Hg. Cardiac ischemia defined by S-T depression > 0.1 mV on a 12 lead EKG taken at rest.
Kanevskaia, Zh. S., Maksimova, L.I., Kublanova, P.S., Shevyreva, N.A., Sineva, E.L., Markova, T.S., 1977	Cross-sectional	Industry, Russia	Males and females (35-39%) aged 25-45 yrs. Group 1 = 256 workers exposed to stable noise; Group 2 = 284 exposed to pulsed noise; Group 3 = controls not exposed to noise exceeding the Maximum Permissible Level. No vibration in work area.	Group 1 = 90-100 dBA steady noise; Group 2 = 107-117 dBA pulsed noise; Group 3 = levels not given; 60% of workers employed ≥ 10 yrs. No information on sources, instrumentation measurement or subjects.	Blood pressure; urine adrenalin and noradrenalin. No information provided on diagnostic criteria, or measurement. (Other measures were symptoms, EEG responses, hearing loss, skin vibrational sensitivity.)
Kobets, G.P., 1972	Cross-sectional	Industry, Russia	Multiple groups: 446 women employed in warehouse & storage work; 390 practically healthy workers; 147 female concrete workers; 144 women subject to the effect of noise; 89 persons suffering from noise disease; similar on mean ages with average length of employment from 7 to 14 yrs.	Noise sources not specified; noise parameters presented for women subject to noise and suffering from noise diseases: 112-122 dB predominately in 1200-1800 Hz range.	Disorders of blood pressure; functional disorders of the heart; temporary work disability.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Authors state some evidence of selection in that to the degree of appearance of coronary complaints, some women changed or quit their jobs and so escaped the study.	Hypertension Prevalence Ratio for Group III (high duration) to Group I (low duration) was = 1.8; for Group II to Group I = 1.5; for Group III to II = 1.3.	Chi-Square.	The frequency of hypertension is significantly greater, the longer the occupational exposure to noise. There were no significant differences between subject groups as to "tendency toward inadequate blood supply" in the EKG nor were pain complaints associated with these changes. Drop of the S-T section greater than 0.2 mV was significantly more frequent in Group II than in Group I (but no differences between Groups II and III) with complaints typical of coronary disease accompanying the changes. The frequency of coronary disease in women with hypertension was 6 times greater than in persons with normal blood pressure. "The studies show that many years of work under noise significantly affect the frequency of the occurrence of arterial hypertension and symptoms of inadequate blood supply of the heart muscles in the EKG. The frequency of these disturbances is greater the longer the occupational exposure to noise."
Selection and measurement bias possible. No evidence of statistically controlling for any variable.	Not stated.	No statistical tests given.	"Therefore, the continuous exposure of the organism of the worker to industrial noise can be compared to chronic acoustic stress capable of causing various functional cerebral-visceral disorders."..."It can be assumed that pulsed noise as well as stable noise provokes the stimulation of the adrenaline ring."..."Undoubtedly, the degree of these disorders is related to the character of noise, its intensity, duration, acoustic effect, the functional state of the central nervous system, and the individual sensitivity of the organism to noise irritants."
Selection bias; measurement bias; no evidence of controlling for confounders.	Not stated.	No statistical methods given; no data presented.	"While it can be fully confirmed that the specific disease and disorders (arterial hypertension or hypotension, functional disorders of heart action, neurosis) are consequences to a certain degree of the specific effects of the named hazards, the injuries of the upper respiratory passages, chronic gastritis and ulcers, and cholangiohepatitis can hardly be traced to their specific effect." The index of cases of the sick rate (total data) was lowest in the control group, rose 2.5 times in workers subject to the effect of noise, increased 3.9 times with development of noise illness, 3.2 times in healthy concrete workers and 3.9 times in individuals with vibration disease.

Industrial Noise: Some Adverse Effects Reported by the Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Liubashevskaia, Z.A., Solonin, IU. G., 1976	Before-after improvements in work area; apparently not the same workers.	Industry, (Forging & pressing shops) Russia	28 workers 22-54 yrs. of age before improvements; 30 workers after improvements.	Before improvements general noise level: 95 dB average and 78-120 dB ranges; after 3 yr. improvement: 90 dB average and 71-110 dB range.	Blood pressure; no description and no data provided.
Pokrovskii, N.N., 1966	Cross-sectional	Industry, (Machine-building) Russia	99[illegible] fitter & lathe operators, mean age 17-55 yrs.	Group 1: 408 exposed to medium frequency stable noise at 80-85 dB; Group 2: 587 workers exposed to high frequency pulsed noise at 90-95 dB.	Blood pressure taken during first 2-3 hrs. of AM shift, after 20 min. rest, in plant clinics; classified by Rosin as hypotension, diastolic hypotension, systolic hypertension, decapitated hypertension and hypertonic condition. EKG readings on 71 workers exposed to 80-85 dB noise and 113 workers exposed at 90-95 dB.
Shatalov, N.N., Saitanov, A.O., Glotova, K.V., 1962(a)	Cross-sectional; before & after work.	Industry, (Ball-bearing & twisting shop) Russia	156 workers in twisting plant; 144 workers in ball-bearing plant.	Twisting shop: 85-95 dB; ball-bearing shop: 114-120 dB; mixed medium and high frequencies in both shops. Duration of exposure - 143 persons had worked 10 yrs. or more.	Blood pressure by Korotkov method; high BP > 130/90 mm Hg for persons < 40 yrs. of age and 140/90 for persons > 40 yrs.; ECG, oscillography, ballisto-cardiography. No data on measurement procedures or conditions of testing.
Shatalov, N.N., Ostapkovich, V.E., Panomareva, N.I. 1969(c)	Cross-sectional	Industry, (Ball-bearing plant) Russia	806 persons exposed to noise; 210 men & women of respective ages with normal hearing, not exposed to noise.	General noise level 90-122 dB, high frequency.	Blood pressure readings taken in AM before work. No definitions or criteria given.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Apparently no confounders considered; potential for many internal validity and selection problems; apparently different group of workers before and after improvements.	Not stated.	None stated.	"Arterial pressure (systolic and diastolic) had a decreasing tendency at the end of the shift...Repeated physiological examinations in 15 forge workers and 15 apprentices showed a decrease in the functional strain upon the organism...The measures implemented were effective from the economic and physiological-hygienic point of view, and can be recommended for other plants."
Selection bias, nonresponse and measurement bias. Age partially controlled; no evidence of control for family history, kidney disease, obesity exercise, medications, etc.	Not stated.	Test according to Fisher.	"Under the influence of intense industrial noise, the arterial pressure of workers can change in relation to individual characteristics in both directions: toward its increase, and toward its decrease as well. In persons exposed to the systematic effect of intense noise, blood pressure is characterized by more pronounced oscillations."
Selection bias; no evidence of controlling for any major potential confounders in the analysis. Age may have been partially controlled, sex not considered.	Not stated.	Data given in % of subjects with defined health index; no statistical tests.	"Very often the workers examined had labile arterial pressure. According to the electrocardiographic data, bradycardia with a tendency toward retardation of the intraventricular conductivity and the falling off of the T-wave were observed, which were more common after physical stress and at the end of the work day. In the group of workers exposed to the effect of noise of greater intensity, functional changes in the cardiovascular system were more common and more pronounced." The hypertensive effect of noise was not observed.
Selection; nonresponse; measurement bias. Stratified by age of < 40 yrs. and > 40 yrs. No adjustment for obesity, job stress, co-morbidities.	Not stated.	Means, S.D., t-values.	"The comparison of the state of hearing and arterial pressure in persons working in conditions of the effect of intense industrial noise showed that the changes of the arterial pressure precede hearing damage. Therefore, one cannot exclude the role of vascular disorders in the development of occupational hearing impairment. In those cases when hearing is already lowered there is no further progression of the processes of sharp dependence on the state of the arterial pressure."

Industrial Noise: Some Adverse Effects Reported by the Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Shatalov, N.N., Thurov, M.A., 1970(d)	Cross-sectional	Industry, (Ball-bearing plant) Russia	2034 men, 1896 women, 20-59 yrs. of age, grouped by noise exposure: 1) 1275 fitters, loaders, lathe operators; 2) 339 operator testers of cranes; 3) 1172 scientists; 4) 1144 lab technicians & skilled mechanics.	Group 1: broad band noise at 95-112 dB with high frequencies dominating; Group 2: noise analogous to Group 1 plus tension; Group 3: no noise (implied); Group 4: control; work not connected with noise. No additional information provided.	Systolic and diastolic blood pressure; hypertensive disease; no definitions or criteria stated.
Suvorov, G.A., Denisov, E.I., Ovakinov, V.C., Tavtin, IU.K., 1979	Cross-sectional	Industry, (Machine building) Russia	740 workers exposed to high noise divided into 4 groups and 121 workers exposed to low noise of about 70 dBA. 587 males and 274 females; average age 34-38 years.	Group 1 = 121 workers at 70 dBA noise level; Group 2 = 139 at 84 dBA; Group 3 = 168 at 93 dBA; Group 4 = 267 at 100 dBA; Group 5 = 166 at 115 dBA; average length of employment 11-16 years.	Hypertension defined as BP > 140/90 mm Hg; hypertensive disease as BP 159/94 mm Hg; hypotension as BP < 100/60 mm Hg; neurocirculatory asthenia by medical specialist Dx.
Troianskii, M.P., Sidortsov, I.P., Petrova-Golubenko, L.B., 1971	Before-during-after work comparisons on a cross-sectional group of workers.	Industry, (Diesel & blower specialists) Russia	55 workers; Group 1: 16 working 12 hrs. with 24 hr. breaks; Group 2: 24 working with 12 hr. breaks; Group 3: 15 working under same conditions without noise (control group).	Noise range of 94-97 dBA in medium and high frequencies.	Blood serum cholinesterase by Hestrin's method; arterial blood pressure; no additional information provided.
Zvereva, G.S., Ratner, M.V., Kolganov, 1975(b)	Cross-sectional	Industry, (Pipe & sheet rolling) Russia	340 workers; no age given; not categorized by exposure levels.	4 areas of the plant described; 1 = 122 dB; 2 = no data; 3 = 115 dB; 4 = up to 142 dB; no use of dosimeters; duration of service given as < 1 yr., 1-2 yrs., 3-5 yrs., 6-9 yrs. and 10 yrs. or more.	Arterial pressure; pulse rate; complaints. No quantitative data provided; no diagnostic criteria or measurement information.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Selection bias, measurement bias, age and sex controlled by stratification and standardization procedures. No controlling evident for other potential confounders such as obesity, exercise, medications taken. Hearing thresholds not obtained. Duration of employment unknown.	Not stated; calculated from table: Age-adjusted (male) prevalence ratio of hypertension for noise exposed plus tension group compared to control group 3.4.	t-tests for mean differences; tests for rates not stated.	"The studies conducted show that the exposure to industrial noise causes an increase of systolic pressure (in the age groups below 40 years) and a rise of incidence of hypertensive disease, compared to the control. Given the intensity of the effect on the incidence of hypertensive disease, exposure to noise as an industrial factor is sufficient cause for neuropsychic tension. The combined effect of the two industrial factors mentioned increases the number of pressure reactions to an even greater degree, in connection with which the incidence of hypertensive disease is significantly augmented."
Selection and non-response bias; incomplete control of age. No controlling for sex, weight, history of disease.	Increase in neurovascular impairment of 0.5% with each 1 dBA increase in noise level.	Regression.	"...At low noise levels, neurovascular disorders prevail, while hearing losses prevail at high levels. Qualitative and quantitative dose-response relationships were established in respect to both the hearing function and mediated responses: the rate of increase in hearing and neurovascular impairments depended on the level of noise, and was 1.5% and 0.5% respectively, per 1 dBA of increase in the noise level; the increased noise level accelerates the symptoms of non-specific mediated impairments (on the average by 13 years with the noise level increased by 31 dBA), most of which belong to hypertonic states."
Selection, no controlling for any variables such as age, sex, length of employment.	Not stated.	Means, S.D., p values; no specific tests noted.	"In specialists working under exposure to the effect of acoustic noise, during examinations immediately at the place of work, statistically significant lowering of the activeness of cholinesterase in blood, extension of the latent period of the dermographic reaction, and a clearer retardation of the pulse during Oschner's test, with a maximum definiteness at the end of the shift, were observed. After the end of the effects of noise, the activeness of cholinesterase was restored. With 12-hour shifts, full restoration occurred after 24 hours. The level of the lowering and the time of restoration of the activeness of cholinesterase can serve as a criterion for the evaluation of the harmfulness of the effect of noise and the character of the response reaction of the organism."
Selection, non-response bias; no obvious control group.	Not stated.	None stated.	"One-hundred sixty-eight persons (50%)...were found to have hearing impairment. ...changes in the neurological state were found in many workers...Complaints in many cases were combined with objective symptoms: disorders of arterial pressure (more often elevated pressure), pulse lability, decrease of vibration and pain sensitivity (up to full anesthesia), thermal asymetry, trembling of arms and eyelids, instability in the Romberg position."

Table A-5
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
English Literature

Industrial Noise: No Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Brown, J.E., Thompson, R.N. Folk, E.D., 1975	Historical prospective/ Cross-sectional	Airline industry, U.S.A.	29 pilots 29 non-flying executives	:8 hour DRC (90dBA) contour exceeded or equalled in all aircraft except jet transports. :Aircraft cockpits. :Duration - 6000 hours of flying time or more. :No noise levels given for executives.	Heart Rate, SBP, DBP and Cholesterol level from annual health records.
Cohen, A., Taylor, W., Tubbs, R., 1980a	Cross-sectional	Industry: paper-making plant, U.S.	51 workers with hearing loss. 51 workers without hearing problems	Proxy measure-high frequency noise induced hearing loss at 65 dB or more at 3000, 4000 and 6000 Hz. Noise intensity levels varied: paper machines at 98-102 dBA and cutting-sorting-machines at 85-92dBA; duration of work exposure: 22.3 yrs. for hearing loss group and 12.5 yrs. for controls.	BP - based on last 2 of 3 readings, taken in recumbent position after 30 minutes rest, by trained technicians unaware of hearing status, included first systolic phase and phase IV and V diastolic. Hypertension defined by WHO criteria of > 160/95. Pre-existing history and medications recorded.
Lees, R.E.M., Smith, C.S., Wetherall, L.D., 1980b	Historical prospective, paired cohort	Industry, Canada	70 pairs of workers.	High noise = > 90 dBA for a minimum of 3 consecutive years: low level < 85 dBA for total work history in plant. Duration: 8 hour shifts, 30 pairs exposed 3-6 years. 22 pairs exposed 7-20 years. 18 pairs exposed 11-15 years.	Counts of new events of IHD, hypertension, M.I. No diagnostic criteria or baseline data shown.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Hearing thresholds monitored annually. Comparability of pilots and executives not given. No statistical controlling.. Selection bias not addressed.	Not stated	t-test to compare groups; F-test to evaluate degree of change over time.	"No changes due to noise exposure were observed in BP, heart rate, cholesterol, or glucose levels."
Analysis controlled for age and body size. No controls noted for race, sex, comorbidity, temperature, etc.	Not Stated	Covariate analysis; no other statistical tests given.	"Statistical evaluation of the blood-pressure data found no reliable difference between the worker groups with high-frequency loss versus normal hearing even with covariate analysis designed to adjust for differences in age and body size (weight/height ratio)...Clearly, it is too early to draw any conclusions about noise as a causal factor in cardiovascular disease."
70 pairs matched on age, exposure period, duration of employment; 36 persons could not be matched; no attrition noted. No data on hearing thresholds. Work shift controlled in analysis.	Not stated	Univariate methods; Pearson's correlation; Chi-square; one way ANOVA.	"There were no significant differences between the 2 groups for both the exposure periods and total study period in incidence of new medical conditions."

Industrial Noise: No Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Lees, R.E.M., Roberts, H.J., 1979a	Cross-sectional	Industry, Canada	62 with noise-induced hearing loss; 62 controls	Hearing loss used a surrogate for noise exposure - cases with hearing loss > 30dB at 4000 Hz; control group of 62 drawn at random from quiet areas. Noisy areas = 95-98dBA. Duration of exposure not given. Audiograms after 12 hours out of noisy area.	BP after 7 min. rest, taken "blind". Hypertension defined as ≥ 140/90mm. Hg.
Malchaire, J.B., Mullier, M., 1979	Cross-sectional	Industry, Belgium	1030 car assembly line; 581 wire mill workers. 501 controls. Males.	Intensity = 92 to 100dB(A) for assembly line; 93 to 97 dB(A) for wire mill with average equivalent noise level = 95 +dB(A); no information on dosimeter instrumentation procedure, duration of exposure. Audiometric tests performed over 3-4 year period.	BP taken by occup. MD: Criteria for determining DBP reading not stated; pre- or post-work shift not stated.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
(Stratified by age and random sample drawn) - matched by age and duration of employment. No other variable considered.	Not stated	Means, S.D.	"No evidence of a relation between increased SBP or DBP and hearing loss"
Control of age and hearing deficit by stratification - controls lacked data on hearing; excluded males with known CVD; all of similar social class.	Not stated	27 Chi-square tests with one statistically significant.	No relationship between noise and BP observed.

Table A-6
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
English Literature
Transportation Noise, Neighborhood Noise and Community Surveys: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Cohen, S., Evans, G.W., Krantz, D.S., Stokols, D., 1980b	Cross-sectional	Community Survey - schools U.S.	262 children in 3rd & 4th grades; 142 from noisy schools; 120 from quiet schools	Aircraft overflight noise; mean peak in noisy school = 74 dB; quiet school = 56 dB; highest reading in noisy school = 95 dB; quiet school = 68 dB; airport noise contours to approx. levels outside homes of noise - school children; levels monitored 1 hr. in AM and PM.	Mean of 2nd & 3rd BP readings taken on consecutive days with automatic BP recorder in quiet room; coders "blinded" to test conditions.
Knipschild, P., 1977a	Cross-sectional	Community survey The Netherlands	2233 individuals in high noise area; 3595 in the lesser noise area; ages 35-64.	Aircraft noise measured at center of village, much noise = B = 40-60 where NNI > 37; less noise = B = 20-40 where NNI = 20-37.	No raw data provided. Hypertension defined as SBP > 175 and or DBP > 100; angina; pathological heart shape; pathological ECG.
Knipschild, P., 1977b	Cross-sectional	General practice survey. The Netherlands	PAR = 12,000 in high noise area. PAR = 17,500 in low noise area.	Aircraft flying over area. 3 areas exposed: E_1: B = 45-55 E_2: B = 35-45 NNI 33 - 50 EC: B = 20-35 NNI = 20-33; Control area B = < 20 or NNI < 20. Village exposed from 1968-1974 indirect line of runway.	Dx. of CVD from general practitioners. Patients seen by different GP's in the areas. No definition of hypertension.
Meechan, W.C., Shaw, N., 1979	Cross-sectional	Neighborhood; airport noise; mortality data U.S.	86,200 in test area; 77,968 in control area.	Aircraft, jet engine noise. Intensity: within 90 dBA + noise contour for test area; 45-50 dBA for control area. No further information provided.	Stroke deaths from county mortality files.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Ecologic fallacy; self-selection; suspicion bias not controlled - home noise contours not available for quiet homes; group matching and analysis controlled for hearing, grade, race, SES, no. of children, but not age of child.	Regression coefficients not reported	Regression analysis	"Children from noisy schools have higher BP and are more likely to give up on a task than children from quiet schools...The greatest differences between the noise and quiet groups occurred during the first 2 years of exposure."
Less than 50% of adults responded with 39% in high noise area and 43% in lesser noise area responding. No evidence of controlling for confounding. Ecologic fallacy possible.	Regression line: % hypertension = 0, 34 B - 0.57 (for each 3 unit increase in B, there is 1% increase in hypertensives.)	Cochran - 2x2 contingency tables.	"In areas with much aircraft noise ($B > 40$) the prevalence of CVD appears to be higher."
Age and sex controlled; no other variables considered; villages differed in socioeconomic class; probable self-selection into serviced.	Not stated	Cochran - 2x2 tables.	For persons aged 15-64 it was found that the contact rate in the exposed area was almost twice that in the non-exposed area. For 15-64 yr. olds, the contact rate for CVD was 9% in E_1, 6% in E_2 and 5% in area C.
Two areas matched on age, income, racial breakdown; data presented to show areas actually differed on these variables. No controlling. Reporting errors differend in the 2 areas. Ecologic fallacy possible.	Not stated	Chi-square to test differences in deaths between test and control areas.	"There has been an increase in mortality rates from stroke for the area of heavy noise radiation under landing jet aircraft as compared with an area removed from such noise effects."

Table A-7
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
English Literature
Transportation Noise, Neighborhood Noise and Community Surveys: Some Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Cohen, S., Evans, G.W., Krantz, D.S., Stokols, D., Kelly, S., 1981c	Cross-sectional/ longitudinal design	Community survey - schools U.S.	262 children in 3rd & 4th grades; 142 from noisy-schools; 120 from quiet-schools; at 1 yr. follow-up: 80 from quiet; 83 from noisy schools with 44 in noise at both testings and 39 in noise abated rooms at 2nd testing	Aircraft overflight noise levels in classrooms at T_1: Noisy = 79.06 dB Abated = 63.17 dB Quiet = 56.60 dB at T_2: noisy = 70.29 L_{eq}; 55.82 L_{33}; 91.50 Peak dB(A) abated = 72.92 L_{eq}; 49.27 L_{33}; 71.27 Peak dB(A) 300 overflights/day; 1 flight every 2½ min.	Mean of 2nd & 3rd FP readings taken on consecutive days with automatic BP recorder in quiet room; coders "blinded" to test conditions.
di Cantogno, L.V., Dallerba, R., Teagno, P.S., Cocola, L., 1976	Quasi-experimental	Laboratory -- "silent" booth Italy	33 males exposed to noise: 11 normal; 11 diabetics (male) 11 coronaropathic; 11 normal males were controls	Short-term: 10 one minute recordings of traffic noise. Intensity: L_m dB 88.8 L_{eq} dB 89.4 L_m dB(A) 71.6 L_{eq} dB(A) 73.1 Administered via headphones; Uher 4400 Stero Report 1C recorded fed noise through amplaid 500 audiometer; amplifon G5 Silent booth erected in a room lined with soundproof panels.	BP automatically taken every min. with Erka Diasist Apparatus; ECG and polygraphic data using standard techniques.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Sampling attrition bias. Noisy-school children who were not retested had higher BP than those retested whereas being retested was unrelated to BP for quiet-school children. Self-selection; ecologic fallacy; group matching and regression analysis controlled for hearing, grade, race, SES, no. of children, but not age of child.	Regression coefficients not reported.	Regression analysis.	Cross-sectional sample at T_1 "indicated inflated SBP and DBP for noisy-school children". There were no effects of noise in the longitudinal data which was as expected since a relatively high proportion of noisy-school children with high BP were lost to attrition.
Control group younger than comparisons: controls $\bar{x}$ = 27 years normal subjects $\bar{x}$ = 36.7 coronaropathic $\bar{x}$ = 52.0 diabetics $\bar{x}$ = 46.4 yrs.	Not stated	Student's interval estimation.	Uric acid increased with noise exposure, esp. in dysmetabolics; blood cholesterol values increased; product of cardiac frequency and systolic arterial pressure tended to fall during acoustic stimulation in normal subjects, but stayed high in dysmetabolic group. "Road traffic noise may be responsible for an increase in myocardial energy requirements."

Transportation Noise, Neighborhood Noise and Community Surveys:
Some Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Graeven, D.B., 1974	Cross-sectional	Community survey U.S.	Quota sample of 20% of females; 552 with 169 from Area I (highest noise) Area II = 96. Area III = 98. Area IV = 88. Control area = 101.	Airplane noise measured by Noise Exposure Forecase (NEF): Area I = NEF > 40 II = NEF = 35-40 III = NEF = 30-35 IV = NEF < 30. Average of 30 planes per hour during day and 8 per hour during night. Control area = not within the flight path of a major airport.	Self-administered Symptom Check List
Mosskov, J.I., Ettema, J.H., (II) & (IV), 1977a & c	Lab experiments with aircraft and traffic noise	Controlled laboratory The Netherlands	12 healthy males (different subjects each noise source) 19-26 yrs. old; served as own controls.	Aircraft: L_{eq} 84-91 dB(A); Traffic: L_{eq} 83.5 dB(A); Total duration of noise 30-40 sec.; highest intensity for 6-10 sec. Taped noise presented through headphones in soundproof room. Three 15 minute exposures with 2 rest periods of 5 minutes between.	BP, pulse rate, respiratory rate.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Age, family income, education, length of residency in community, length of time in the home were considered. Control city respondents were older than noise exposed subjects.	Y = -.03 (zero-order) between level of exposure and number of health problems. Multiple R = .198.	Stepwide Regression for total sample and 5 separate regressions for the different levels of exposure.	"Exposure to airplane noise was the 3rd most important factor in determining health problems." (Awareness and annoyance reactions were most important.)
Random assignment of noise type not performed; random assignment of noise, mental load, combined load within each experiment.	Not stated	Wilcoxon Test.	Exposure to aircraft and traffic noise caused an increase in DBP, and decrease of pulse pressure and HR RR.

Table A-8
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
Translated Literature

Transportation, Neighborhood and Community Noise: Some Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Koszarny, Z., Maziarka, S., Szata, W., 1965	Cross-sectional	Neighborhood survey (airport & residential noise) Poland	256 in high noise zone; 255 in low noise zone; men and women age 20-70 yrs.	High noise zone = > 100 dB(A); low noise zone = 80-90 dB(A); no other information provided.	Frequency of symptoms including cardiac pains and taking of cardiac medicines.
Meinhart, P., Renker, U., 1970	Cross-sectional	Industry, Germany	807 males with noise impaired hearing compared to 3948 selected to be representative of men seen in health clinic in the same district.	Noise from industrial area of Halle District; no information as to noise parameters and no hearing-impairment criteria given.	All circulatory diseases, functional heart disease, myocardium injury, hypertension, hypotension, peripherial circulatory problems. Survey and clinic record data. No diagnostic criteria and no definitions given.
Von Eiff, A.W., Neus, H., 1980	Cross-sectional; random survey.	Community survey, (traffic noise) Bonn, Germany	458 men, 437 women; ages 20-59	High traffic area = 63-73 dBA noise level; Low traffic area = maximum of 50 dB; Exposed for minimum of 3 yrs.	Hypertension and treatment for hypertension determined by questionnaire.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Groups similar on type of education, occupation, working conditions, age & sex but differed on housing and living conditions - poorer housing in low noise area. Data stratified by age and sex. No other evidence of control.	Not stated.	Chi-Square and t-test.	"The relationship between some symptoms of aggravation and ill health and the acoustic conditions in the place of residence indicates the probability of the negative influence of airport noise on the residents' state of health." The high percentage of persons complaining of symptoms and the frequent occurrence of symptoms of illness among women, but not men, from the worst acoustic conditions seems to support the relationship between airport noise and the state of health. "On the other hand, one cannot exclude the assumption that the above mentioned indexes result from other unfavorable effects of the environment, especially the work environment."
Ecologic fallacy; selection bias; age partially controlled by stratification; no evidence of control for comorbidities, treatment or other variables.	Prevalence ratios implied for noise injured vs. controls: Myocardial injury = 2.0 Hypertension: 12-40 yrs. old = 7.6, 40-65 yrs. old = 9.7, 65+ yrs. old = 8.2.	Data presented as proportions for comparison; statistical tests apparently applied, but not described.	The prevalence of myocardial injuries was about twice as high for the noise-injured group as the controls; hypertension was higher among noise-impaired at all ages except the 65 and older; for all age classes, the frequency of heart circulatory diseases and especially hypertension and hypotension began rising after five years employment and rose precipitously after 20 years work; no differences between the groups were observed for functional heart-circulatory diseases or peripheral perfusion disturbances. "The steady noise influence is an important factor of the origin of heart-circulatory diseases."
Ecologic fallacy; measurement bias.	Prevalence ratio of 1.6 for hypertension in high noise area compared to the low.	p values given; data given as proportions; no statistical test stated.	"Noise is very troublesome on streets with high volume traffic. Specific high blood pressure as a treated disease was mentioned significantly more often in the high noise area than in the low noise area."..."The data on men between the ages of 20 and 39 who lived in the loud-noise area revealed hypertensive treatment to be dependent upon length of residence. This was not the the case in the low-noise area."..."The results of the investigation justified a prospective, interdisciplinary, epidemiological study, in which the physical measurements are correlated with the measured blood pressure data."

Table A-9

Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System

English Literature

Transportation Noise, Neighborhood Noise and Community Surveys: No Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Drettner, B., Hedstrand, H., Klockhoff, I., Svedberg, A., 1975	Cross-sectional	Community Survey Sweden	100 males; age 30 yrs; 388 reported history of noise exposure 374 no exposure	Questionnaire used to determine exposure; amount of hearing loss expressed as the sum of dB hearing loss of both ears at 5 frequencies - levels 1000-6000 Hz.	Heart rate, BP measured after 10 min. rest in supine position; cholesterol, trigycerides.
Frerichs, R.R., Beeman, B.L., Coulson, A.H., 1980	Vital Records Mortality Study; cross-sectional	Neighborhood; airport noise; mortality data U.S.	1970 census: population of 89,019 in test area; 77,611 control area	Test area within the 90 dBA or more noise contour of airport; control area with noise level of 45-50 dBA.	Age, race, sex and cause of death-specific-mortality rates. CVD: ICDA 390-448 and ICDA 430-438 as coded by the state of California; limited to specific study areas.
Hedstrand, H., Drettner, B., Klockhoff, I., Svedberg, A., 1977	Cross-sectional	Community survey Sweden	393 males with noise induced hearing loss; 376 males with normal audiagrams.	Surrogate measure-noise-induced hearing loss defined as $\geq$ 65dB at 3, 4, or 6K Hz and normal tone audiograms as < 20dB at all frequencies.	BP in supine position hypertension defined as $\geq$ 160/100 mm. Hg.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Non-response bias - only 83.9% participation rate; self selection and selective recall; potential for misrepresentation of data with the repeated multiple correlations of all variables.	Not stated	Chi-square; K-S test; correlations between and among 10 risk variables and hearing loss.	No significant correlations between hearing loss and 10 risk factors except for smoking habits - factors included SBP, DBP, heart rate. "Among 92 who had smoked more than 10 cigarettes daily, but had not been exposed to noise, the amount of R-sided hearing loss was greater than in 105 individuals who had never smoked and had not been exposed to noise."
Ecologic fallacy; errors in mortality data such as diagnosis, reporting; self selection into test and control areas. Data were adjusted for age, race, sex.	Not stated	SMR; direct and indirect age adjustment.	"We were unable to validate the findings of Meecham and Shaw... Once the confounding effects of age, race and sex were taken into account by direct and indirect methods of standardization, there was little difference in the mortality experience of the airport and control areas. Adjusted mortality rates due to all causes, cardiovascular diseases, or cerebrovascular disease did not differ appreciably between the two areas...Clearly any link between airport noise and mortality must be based on sounder evidence than has been presented to date if causality is to be inferred."
Age and sex controlled. No controlling indicated for years of noise exposure, comorbidities, etc.	Not stated	Means and S.D.	No significant differences in mean BP between hearing loss group and controls.

Transportation Noise, Neighborhood Noise and Community Surveys:
No Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Knipschild, P., Salle, H., 1979c	Cross-sectional	Community with noisy streets in Netherlands.	399 living in noisy areas. 1342 living in quiet areas. Housewives 40-49 years.	Automobile noise, noisy streets L_{eq} = 65-70 dB(A), thus L_{dm} >62.5(A). Quiet streets: L_{eq} = 55-60 dB (A), thus L_{dm} < 62.5 (A)	Consultation with cardiologist. Hypertension SBP $\geq$ 160 and DBP $\geq$ 105 mm Hg at rest in sitting position; Angina pectoris; Ischemia on ECG by Minnesota Code.
Takala, J., Varke, E., Vaheri, E., Sievers, K., 1977	Cross-sectional	Community survey Finland	32 men exposed to noise and with hearing loss; 67 men with normal hearing	Noise exposure hearing loss defined as thresholds of hearing 65 dB or more at 4000 and 6000 Hz; normals at 20 dB.	Single BP reading, R-arm, sitting for 3-5 mins., 5th phase DBP recorded. Hypertension defined as > 160/100 mm Hg.

Bias and Potential Bias Due To Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings As Reported By Author
Data from screening program; states that attention was paid to possibly confounding factors (age, civil status, financial situation, smoking habits, weight, physical activity); ecologic fallacy; self-selection; response rate differed between noisy and quiet areas.	Not stated	Fisher's Test-one-sided	"This study showed no indication for a relationship between traffic noise and cardiovascular disease."
93% participation rate. No variable, including age, controlled in the analysis. Volunteer and migration bias possible.	Not stated	Student's t-test	No statistically significant differences in SBP nor DBP between the normal hearing and noise defect group.

Table A-10
Summaries of Epidemiologic Studies of the Effects of Noise on the Cardiovascular System
Translated Literature

Laboratory Studies: Adverse Effects Reported by Author(s)

Author & Year	Study Type	Study Site	Sample Size	Noise Exposure Measure-Quality-Long vs. Short Term	Cardiovascular Response Measure-Quality
Burger, F., Klimes, J., 1975	Quasi-experimental; simulated noise of tank motors.	Laboratory Czechoslovakia	20 untrained conscripts, aged 19.3 ± .09 yrs.; 10 exposed to 20 mins. noise while exercising; 10 exposed to 2 hrs. of noise with exercise.	Steady noise of tank motors with a known spectrum recorded and conveyed to room by 2 loudspeakers; short-term noise at 90, 100, 110 dB; long-term noise at 80, 100 dB.	Pulse frequency: EKG curve evaluated with a 1-channel electrocardiograph.
Klotzbuecher, F., 1976	Quasi-experiment	Laboratory Germany	10 healthy male vocational students; 17-19 yrs. of age.	3 test conditions randomly assigned to subjects: wide band (white) noise at (1) 55 dB(AI); (2) 70 dB(AI); (3) 85 dB(AI); 3 test groupings: (1) rest, 10 min. sitting; (2) 3 min. rest with noise; (3) 90 min. arithmetic tasks with noise; Tests conducted over a 12 day period.	Heart rate measured by EKG; respiratory rate by thermister; catecholamines determined by method of Euler and Floding.
Mariniako, A., Lipovoi, V., 1975	Quasi-experiment	Laboratory Russia	20 healthy males; 20 observations in each of 4 conditions of noise.	Series 1: 110 dB low frequency noise; steady. Series 2: 110 dB low frequency noise, intermittent. Series 3: 105 dB high frequency. Series 4: 105 high frequency; intermittent.	Tone of the vessels and pulse rate.
Quaas, M., Geiller, W., Platzbecker, I.U., Zoellner, G., 1970	Quasi-experimental	Laboratory Germany	8 clinically & otologically healthy males, 22-35 yrs. of age.	3 test periods: Test 1: wore ear plugs; 10 min. rest; 30 min. continuous exercise; 10 min. rest. Test 2: wore ear plugs; 10 min. rest; 30 min. continuous exercise under 90 dB wide band noise; Test 3: did not wear ear plugs; 10 min. rest; 30 min. continuous exercise under 75 dB wide band noise; 10 min. rest.	Heart rate, oxygen consumption, carbon-dioxide output. Instrumentation described.

Bias and Potential Bias Due to Confounding	Effect Parameter	Analysis and Statistics	Summary of Findings as Reported by Author
Selection bias; Latin square design used to avoid habituation; no other evidence of controlling for confounding.	Not stated.	Test of pairs.	"Even though our experiments allow the conclusion that a decrease of physical ability does not occur, it is, in view of its effects upon the circulatory system, impossible to affirm unconditionally that exposure to noise is completely harmless to the organism."..."but that its negative effects will become manifest after an exposure lasting a number of years. The use of protectors against noise does not prevent its effects upon the circulatory system."
Sequence of test series was random; subjects not randomly selected; no data as to comparability on work exposure, obesity, exercise, etc.	Not stated.	t-test for pairs correlation coefficients.	"The number of significant correlations between performance and physiological reactions decreased a lot more from noise level 55 dB(AI) to noise level 70 dB(AI) than from noise level 70 dB(AI) to 85 dB(AI)"..."The rise of adrenalin secretion under noise strain and mental work found by us is twice as high as 55 dB(AI)-as with noise strain."..."The recommended noise level at concentrated mental work should therefore be lower than 55 dB(AI)."
Selection bias; no randomization described.	Not stated.	No tests indicated.	"A rather noticeable difference in the effects of stable and intermittent noise (especially with high frequency compositions) was noticed in the study of the tone of the vessels."..."After one hour of effects of stable and intermittent noises, a tendency toward decline in pulse frequency, on the average of 2-3 per minute, was observed."
Subjects used as own controls - very small sample (7).	Not stated.	t-test F-test	"The use of ear protectors does not prevent influence of 90 dB wide band noise upon the heart-circulatory system." Heart rate increased under noise conditions.